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JULY 1954
VOLUME 60 NUMBER 1

IN TWO SECTIONS—SECTION 1

Published Monthly by
AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of March 3, 1879. Annual Subscription, $12.00

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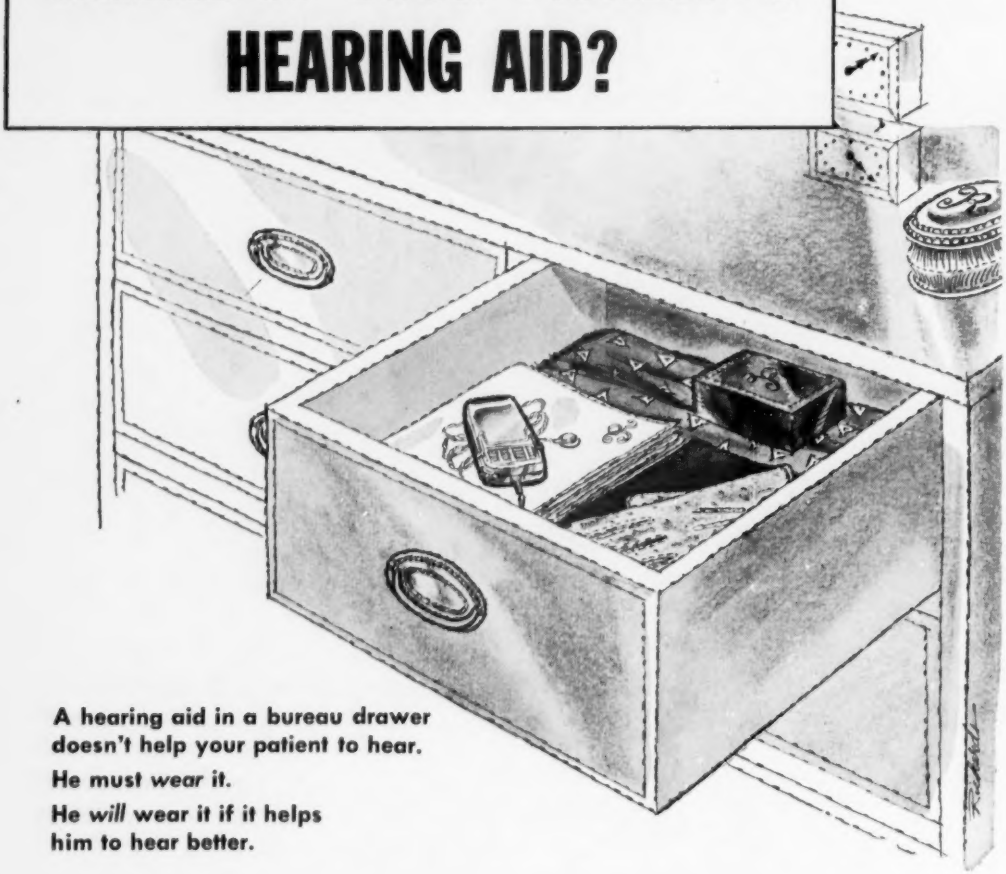

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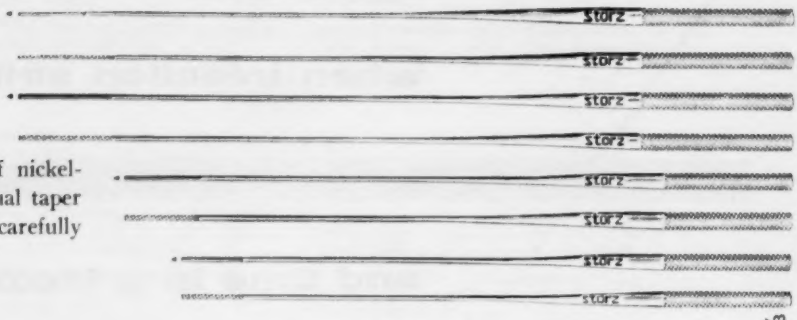

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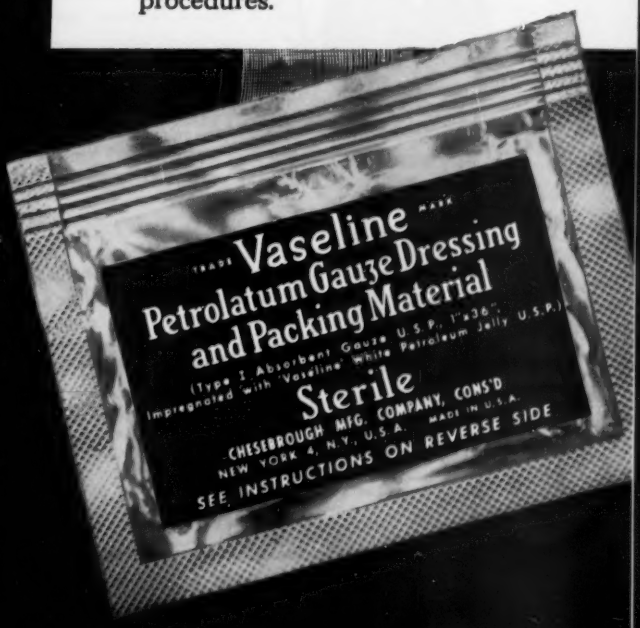
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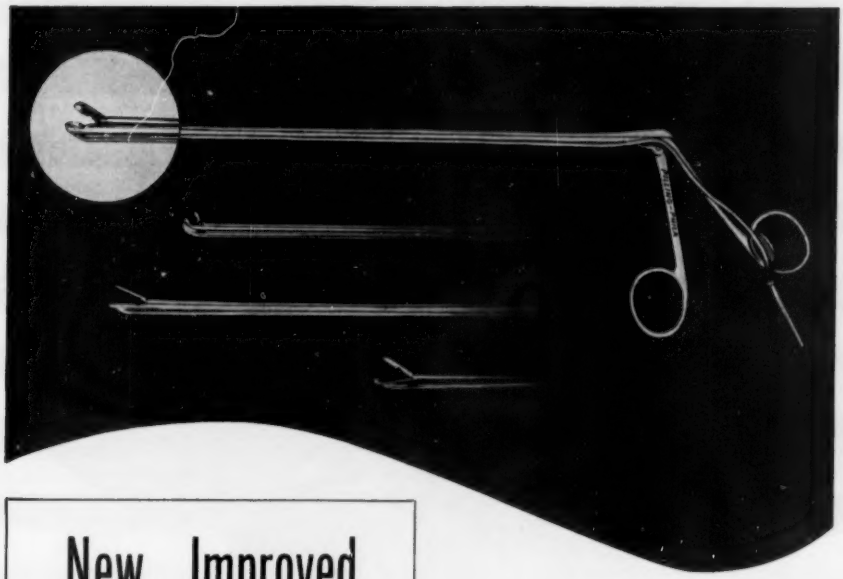

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EARLY TRACHEOTOMY IN SECRETIONAL ANOXIA

RONALD W. TAYLOR, M.D.
VANCOUVER, B. C., CANADA

TRACHEOTOMY has been done since the Middle Ages, usually as a last resort and only for obstruction of the upper respiratory tract. Lee, Tucker, and Clerf [1] (1928) showed that postoperative pneumonias were the result of respiratory obstruction, with consequent atelectasis. Early promotion of coughing and early bronchoscopic suction relieved the obstruction. Durand [2] (1929) demonstrated the effectiveness of postural drainage in relieving pulmonary obstruction in poliomyelitis. The idea of a lower respiratory obstruction, which is the basis of secretional anoxia, was conceived at this time.

Wilson [3] (1931) mentioned tracheotomy as a means of relieving lower respiratory obstruction. Figi [4] (1934) demonstrated that bronchopneumonia is a common complication of pulmonary obstruction and that death in most cases of lower respiratory obstruction was due to delay in performing a tracheotomy. Davison [5] (1936) mentioned tracheotomy for the relief of pulmonary obstruction in poliomyelitis. The actual reporting of results, however, was not done prior to Galloway's [6] work in 1943. During the severe poliomyelitis epidemic in Minneapolis in 1947, Priest, Boies, and Goltz [7] added considerable knowledge with exhaustive studies of several hundred cases. A comprehensive report on this subject was also given by Galloway [8] (1949), with eight indications for tracheotomy in bulbar poliomyelitis. Bower and associates [9] and Miller and Buck,[10] in 1950, emphasized prophylactic or early tracheotomy in poliomyelitis. Harris [11] (1952) reported a marked reduction in mortality of the bulbar and bulbospinal poliomyelitis cases by doing tracheotomy early in the disease. Von Leden [12] (February, 1953) introduced newer concepts of tracheotomy and increased indications for the procedure. At this time, prophylactic tracheotomy is being done to prevent asphyxia and to prevent pulmonary complications from arising.

PHYSIOLOGY OF RESPIRATION

For a better understanding of secretional anoxia, the physiological factors of normal respiration should be briefly explained.

Normal respiration is controlled by nervous centers located in the medulla oblongata. These centers are regulated by the pH of the blood and by the arterial carbon dioxide level. A rise in the carbon dioxide tension of the blood, or a fall of the pH of the blood to a more acid level, stimulates these centers in such a way as

From the Subdepartment of Otolaryngology, Department of Surgery, University of British Columbia Medical School, and Vancouver General Hospital.

Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

to increase pulmonary ventilation and thus blow off the excess carbon dioxide. Conversely, a rise in the pH of the blood or a fall of the carbon dioxide tension decreases pulmonary ventilation, allowing the carbon dioxide tension to rise to a normal level. The normal arterial carbon dioxide tension is 40 mm. Hg, and the normal pH is 7.4. Under normal conditions, the arterial carbon dioxide tension controls the medullary centers, and the arterial oxygen tension is not involved.

There is a secondary regulating mechanism composed of peripheral chemoreceptors, which normally perform a modest role in respiration. They are concerned only with the arterial oxygen tension, and carbon dioxide exerts no effect on them.[13] When pathological situations affect normal respiration, the chemoreceptors, which are located in the aortic and carotid areas, become extremely sensitive to the lowered arterial oxygen tension. Under these conditions, the chemoreceptors become the center of respiratory regulation.[14] The aortic and carotid bodies are not depressed by accumulation of carbon dioxide in the arterial blood nor by opiates or barbiturates, as are the medullary centers. They cause an increase in respiration when the arterial oxygen tension falls below normal and constitute an important part of normal physiology.

LOWER RESPIRATORY PATHOLOGY

In many pathological states the respiratory centers are depressed or rendered functionless. Anoxia occurs as a result of the respiratory depression. The anoxia is enhanced by fluid accumulation in the tracheobronchial tree and alveoli, which forms a barrier to oxygenation of the arterial blood.[15]

In head, face, neck, or chest trauma, anoxia may ensue as a result of bleeding of the parts of the tracheobronchial tree or from edema of the soft parts around the tracheobronchial tree.

PROTECTION OF THE TRACHEOBRONCHIAL TREE

1. There is a normal tussive action in the tracheobronchial tree. Iodized oil (Lipiodol) studies have shown a peristaltic action.[16] The peristalsis acts in such a manner as to propel foreign material out of the chest, commencing in the distal part of the tree and advancing up the trachea. The respiratory centers (according to Henderson [17]) control not only this expulsive mechanism of the tree itself but also the muscular action of the thoracic cage and, indeed, are capable at times of controlling the entire musculature. Depression of the medullary respiratory centers therefore can hinder this tussive effect.

2. The respiratory tract is lined with ciliated epithelium. The normal sweeping action of the cilia propels exudate from the alveolar and bronchial areas to the pharyngeal surface for expulsion. This action is not under the control of the central nervous system but is influenced by chemical factors and by toxins present either locally on the mucosa or in the arterial blood.[16] Proetz [18] has shown that dry air slows the propulsive action of the cilia and destroys them. Cold air hinders ciliary action. Cilia are inhibited also by drugs, such as morphine.

In most respiratory obstructions, a large amount of secretions collect in the tracheobronchial tree, and ciliary action may be overcome by the increase in the mechanical load. Postural drainage will then materially aid in clearing the tree.

3. The reflex act of swallowing is initiated by the action of saliva passing over sensitive areas which are located in the posterior pharyngeal wall and the base of the tongue.* The reflex arc is composed of afferent impulses traveling through the trigeminal, glossopharyngeal, and vagus nerves and efferent impulses traveling through the latter two nerves. If the reflex arc is interrupted, swallowing becomes faulty or impossible, and pharyngeal secretions, which are continuously forming, overflow into the tracheobronchial tree.

4. The cough reflex begins with inspiration followed by a closure of the glottis. The intrathoracic pressure is built up, the glottis opens, and expiration and descent of the thoracic cage occur. The sudden release of intrathoracic pressure expels bronchial secretions and foreign materials a variable distance along the airway, and repeated coughing eliminates the exudate. The afferent impulses through the vagus nerve are conveyed to the brain stem from the sensitive areas located in the larynx, trachea, and bronchi. The most sensitive peripheral areas are in the region of the tracheal bifurcation and the laryngeal mucosa. The reflex may be initiated from the vagal afferent impulses in the lungs themselves or from the pleura, as well as from the tracheobronchial tree.† Efferent impulses are transmitted to the neuromuscular endings in the tracheobronchial tree and to the muscles of respiration.

PHYSIOPATHOLOGY

Anesthesia of the afferent endings or paresis of the higher centers, inclusive of the medullary centers, can interrupt the reflex arc and cause a depression or absence of the cough reflex. The cough reflex may be depressed simultaneously with the loss of the ability to swallow. This situation, in conjunction with a depression of the bronchiolar tussive action, exposes the respiratory tract to the danger of fluid accumulation and stasis, as the ciliary action may be ineffective in disposing of the saliva normally secreted into the pharynx.

Aided by the force of gravity and suction factors in the lower pharynx, the accumulated fluid passes into the tracheobronchial tree when the normal expulsive means are disabled. By the same token, food, vomitus, and foreign materials can be aspirated when the cough and swallowing reflexes are depressed or absent. Respiratory obstruction occurs and secretional anoxia results. The obstructing mechanism consists of accumulated fluid in the alveolar sacs and alveoli, which prevents the normal exchange of oxygen between the alveolar air and the arterial blood. The arterial oxygen is also reduced as a result of alveolar membrane edema in areas of inflammation which surround fluid accumulations. Bronchiolar mucus plugs block alveolar areas, and atelectasis results. These static areas may easily become secondarily infected, as shown by Coryllos and Birnbaum.[19]

CLINICAL PICTURE

Clinically, this alveolar block is detected from the signs and symptoms produced by the resultant anoxia of the cerebral cells. Cyanosis is dependent on too many variables to be relied upon. If present, it is indicative; if not, anoxia may still be present. The most constant sign is an increase in the pulse rate; however, this sign

* Reference 16, p. 480.

† Reference 16, p. 303.

is not always reliable, owing to extraneous factors.[12] Other signs include headache, anxiety, restlessness, apprehension, confusion, lethargy, and, finally, coma. Nervous tissue is extremely susceptible to damage from lack of oxygen. Permanent changes may occur in the nerve cell as a result of anoxia lasting three to eight minutes (Courville [20]). The cerebral cortex cells are the most sensitive and, to a slightly less degree, the medullary centers.[21] Repeated exposure to anoxia produces a cumulative effect, and permanent damage may ensue with even mild anoxia if the arterial oxygen tension is not elevated and maintained.[8] In view of the lack of constant clinical signs, an arterial blood oxygen estimation is the most reliable test for anoxia at this time. This may be accomplished with an oximeter arterial oxygen saturation test (Kubicek and associates [22]). A rapid, accurate estimation of alveolar oxygen and carbon dioxide tensions and a rapid, accurate estimation of the arterial gaseous components would be effective means of diagnosing anoxia, if these tests were available and satisfactory. A pH estimation of the blood would aid in detecting an acidotic state. However, these tests are not readily available for repeated estimation.‡ One must frequently rely upon clinical observations alone for assessment of an anoxic state.

BLOOD CHEMISTRY IN ANOXIA

When the respiratory center is depressed, carbon dioxide accumulates in the arterial blood. The respiratory center no longer responds to an increase in the carbonic acid present. Pulmonary ventilation is decreased, and a fall in the pH of the blood occurs. Acidosis is the result if the respiratory center depression is maintained, because the compensatory mechanism of the alkali reserve of the blood is overcome. The acidosis, in turn, has a depressing effect on the respiratory center, and the anoxic cycle is unbroken. This may progress to coma and respiratory cessation with death.

While the carbon dioxide tension is rising, as above, oxygen is being metabolized; and, as there are no oxygen reserves, the oxygen tension is lowered because of decreased pulmonary ventilation. When the arterial oxygen tension is lowered to dangerous proportions, the peripheral chemoreceptors (Heymans and associate,[23] 1930) assume primary control of respiration.

In the interval, however, before respirations are returned to normal, fluid or foreign bodies may have entered the tracheobronchial tree. This is particularly likely to occur when the protective mechanisms have been depressed, as has been mentioned previously. Saliva, tenacious secretions, or foreign particles may thus obstruct respiration. In the case of crush injuries to the face, neck, and chest, the obstruction may be blood or a blood clot. Inhalations of carbon dioxide or oxygen will not relieve anoxia in this instance, due to the barrier between the gases and the arterial blood.

RELIEF OF SECRETIONAL ANOXIA

The obvious and most effective therapy is to remove the obstruction to respiration. This can be accomplished by postural drainage, bronchoscopic suction, or, finally, tracheotomy suction in cases in which the more conservative procedures have failed.

‡ Pump, K. K.: Personal communication to the author.

Tracheotomy is the most simple and effective procedure to prevent or relieve anoxia in bulbar and bulbospinal poliomyelitis in cases in which the more conservative measures have failed.§ Tracheotomy suction is preferable also whenever continuous suction is necessary. Tracheotomy with easy access to suction is better tolerated by fatigued patients than is repeated bronchoscopic suction.[12]

The effect of tracheobronchial suction on anoxic signs and symptoms is frequently instantaneous and spectacular when secretions can be readily removed through the tracheal opening, and a direct supply of adequately controlled, humidified oxygen can be administered.

This surgical procedure presents very few serious complications and bypasses a large dead space in the upper respiratory tract.

GENERAL INDICATIONS FOR TRACHEOTOMY

The incidence of tracheotomy is increasing. The following pathological states have the potential to develop secretional anoxia and therefore require close observation to prevent the occurrence of this condition. A prophylactic tracheotomy is preferable to a therapeutic one if clinical judgment indicates the necessity. The common denominator will be seen to be a respiratory depression with loss of the protective reflexes of the respiratory tract.

1. Acute laryngotracheobronchitis or foreign bodies in the respiratory tract.[29]

2. Any acute neurological disorder with respiratory embarrassment as a result of interference with the reflexes of respiration. This includes bulbar and bulbospinal poliomyelitis, paralysis following any febrile illness (diphtheria,[30] polyneuritis), and cerebral vascular accidents.

3. Comatose states with developed or impending respiratory embarrassment. These may be due to trauma, to the action of drugs (phenobarbital, morphine),[12] or to cerebral vascular accidents.

4. Injuries of the face or the cervical spine, or crushing injuries of the chest which obstruct the airway or potentially obstruct the airway by edema and hemorrhage. Blast burns affecting the respiratory tract and thermal injuries are particularly suited to tracheotomy.[12]

5. Old and debilitated patients who are exposed to long operative procedures.[31] Prophylactic tracheotomy may be considered, since the procedure adds very little to the morbidity and, by preventing pulmonary and cerebral complications, may lower the mortality.[32]

6. Severe head injuries, with or without coma, in cases in which the medullary respiratory center has been damaged and there is difficulty in swallowing and expectorating secretions.[33] This classification includes lengthy neurosurgery of the head and neck.

7. Toxins and poisons causing convulsions or coma. Toxic systemic states, such as eclampsia and uremia,[12] are involved. Tetanus,[34] botulism,[35] and common poisonings (diphenhydramine [Benadryl], barbiturates) are other examples.

8. Metabolic disorders such as myasthenia gravis.

§ References 6, 8, and 24 to 28.

ADJUNCT THERAPY

Adequate antibiotic therapy will help solve the problem of secondary infection. The electrolyte and fluid balance can be controlled by measuring the intake and output and by blood chemistry studies. Levin-tube feedings and intravenous therapy can be used to restore the sodium-potassium balance. Humidified catheter oxygen is a primary requisite, which should be administered at a rate commensurate with the vital capacity of the patient. Under certain conditions there is a danger of oxygen overloading, which can be as serious as anoxia itself.[36]

ESTABLISHED METHODS OF RESTORATION OF COUGH REFLEX AND NORMAL RESPIRATION

The patient should be instructed to cough early. Frequent turning and early ambulation of postoperative patients is necessary. Shaking a child or direct stimulation of the sensitive areas in the pharynx by a catheter or bronchoscopic examination may be used as cough inducements.

NEWER METHODS OF STIMULATING NORMAL RESPIRATION AND COUGH REFLEXES

In paralysis of the muscles of respiration, a respirator is needed. The more recently developed Emerson demand-respirator is preferable to the Drinker type.[14] The Emerson respirator more closely resembles and synchronizes with normal respiration. A recent innovation for the respirator case with a fixed chest or poor neuromuscular response is the intermittent electrical stimulation of the phrenic nerves. Another promising addition to the respirator case armamentarium is the exsufflator, or cough simulator.[37] The natural act of coughing is simulated by means of rapid evacuation and filling of the respirator through a vacuum blower unit. The rocking-bed method has also been used to aid ventilation. Gravity is utilized to move the diaphragm, the abdominal viscera alternately compressing and then falling away from the chest. The frog (nasopharyngeal) breathing method is being used in convalescence to permit physical therapy over longer periods of time. Dr. Clarence W. Dail, of the Rancho los Amigos Hospital, Los Angeles, developed a method of instructing paralyzed patients to gulp air as a frog does.

REPORT OF CASES

Bulbar Poliomyelitis.—The first case illustrates the effectiveness of an early tracheotomy in this disease.

Case 1.—A 2½-year-old child was first seen May 4, 1953, with a high fever, stiff neck, and a palatal paralysis with excessive amounts of frothy mucus. Tonsillectomy and adenoidectomy had been performed three and a half weeks prior to this examination. The lower intercostals were paralyzed, the breathing was diaphragmatic, and the cough reflex was depressed. The chest was filled with moist rales, and the cerebrospinal-fluid examination showed 51 cells per cc., with 80% polymorphonuclear leucocytes and 20% lymphocytes.

After admission to the hospital, anoxia was imminent. The swallowing reflex was paralyzed, and pharyngeal secretions were overabundant. An emergency tracheotomy was done, and large amounts of mucinous exudate were suctioned at repeated intervals. Respiratory excursions improved with tracheal catheter suction and humidified catheter oxygen, and the swallowing reflex became somewhat more effective. Levin-tube feedings were given. The child developed a very severe attack of chicken pox on the 23d postoperative day, by which time there had been a

noticeable return of the cough reflex and swallowing mechanism. The tracheotomy tube had been stoppered for three days. Examination of the pharynx and chest evidenced a recurrence of an excessive amount of exudate in the tracheobronchial tree, which required regular suction during the acute phase in the next four days. The tracheotomy tube was removed on the 29th day following a satisfactory resolution of the chicken pox.

Comment.—It is interesting to note that a tonsillectomy and adenoidectomy had been performed three and one-half weeks prior to admission for poliomyelitis. An early tracheotomy relieved the secretional anoxia. Abnormal pharyngeal secretions again presented with the severe attack of chicken pox, and these secretions were readily eliminated by tracheobronchial suction through the tracheal opening. Complete recovery was hastened by the early tracheotomy.

Acute Bulbospinal Poliomyelitis.—The largest group of seriously ill poliomyelitis patients is composed of those with the bulbospinal type of disease, requiring a respirator.[11] Galloway and Seifert [8] have pointed out eight indications for tracheotomy based on symptomatology and clinical findings. These indications are well known. The following case history includes many of the indications.

CASE 2.—A 27-year-old man was admitted in a respirator July 21, 1953. Examination revealed overabundant pharyngeal and tracheobronchial secretions and loss of cough and swallowing reflexes. The chest showed little evidence of expansion, and rales were numerous. Paresis of the right shoulder muscles was noted. In spite of repeated catheter suction and humidified catheter oxygen, he showed overanxiety, a rapid pulse, and a temperature of 105 F. Secretional anoxic signs were obvious. A tracheotomy was done within an hour of admission under general anesthesia, with continuous artificial respiration through an intratracheal tube. The incision was made through the second and third tracheal rings down to the intratracheal tube. Large amounts of mucinous exudate were suctioned from the tracheobronchial tree as well as from the mouth. The patient was returned to the respirator. The temperature dropped within the first 48 hours. Intravenous oxytetracycline (Terramycin), intravenous dextrose and sodium chloride solution, and, later, Levin-tube feedings were administered. Signs of respiratory embarrassment disappeared within the first 10 postoperative hours. The tracheobronchial secretions were markedly diminished within seven days, the chest was free of rales, and other anoxic signs had disappeared. The tracheotomy tube was stoppered on the 14th day, when early signs of movement in the lower chest returned. Damage to the respiratory center will require assessment at a later date. A general improvement, however, was evident. There were no residual signs of cerebral anoxia. The patient is out of the respirator from 12 to 14 minutes at the time of writing.

Comment.—This recent case presents the satisfactory improvement in secretional anoxia with early tracheotomy. Repeated catheter suction and bronchoscopic suction were by-passed in favor of tracheotomy for obvious reasons. Tracheotomy by-passed the large oversecreting pharyngeal surface. Ventilation improved almost immediately. Restlessness and anxiety disappeared with the resolution of the respiratory obstruction. Early tracheotomy is most effective in these cases.[11]

Combined Head and Chest Injury.—The development of chest complications in patients with severe head injuries is an established fact.[33] Most frequently this is due to secretional anoxia. The provision of an early tracheotomy in head injury cases in coma is effective in preventing pulmonary complications. The report of Echols and his associates indicates the value of an early tracheotomy in severe head injuries. The following case is that of a combined head and chest injury as a result of a car accident. This type of injury is not an uncommon occurrence.

CASE 3.—On March 13, 1952, a 20-year-old woman suffered severe brain damage as a result of a car accident, after which she remained in a comatose state for 92 days. The pupils were

unequal; bleeding was evident from the nose and right ear. The arm reflexes were flaccid and the legs spastic. The respirations were varied in spite of continuous administration of oxygen by catheter. Early anoxia was present. Skull x-rays revealed a fracture along the floor of the sella turcica and the right lateral wall of the sphenoid sinus into the greater wing of the sphenoid. Several ribs were also fractured, as shown by x-rays.

A tracheotomy was done within 48 hours, and tracheobronchial suction revealed old blood and blood clot and later considerable mucinous exudate. The respiratory rate steadied and improved in character. She exhibited less decerebrate rigidity within 24 hours. Exploratory burr holes showed no evidence of extradural or subdural fluid. The brain tissue appeared normal. Coma persisted for 92 days, and tracheobronchial toilet was maintained after the tracheotomy and probably prevented pulmonary complications. Signs of mental deterioration were present after the return to consciousness.

Comment.—The signs of respiratory obstruction disappeared after the tracheotomy suction, which revealed old blood and blood clot. A tracheobronchial toilet was maintained throughout the continued coma. It is possible that some of the resultant brain damage was due to cerebral anoxia. The possibility of preventing this additional insult to cerebral tissue by anoxia should always be borne in mind. The chance of its occurrence may be prevented by the early use of tracheotomy, to avert the development of secretional anoxia.

Diphenhydramine Poisoning.—Tracheotomy has been invaluable in those comatose cases due to the assimilation of such narcotics as barbiturates and opiates [12] and due to toxins, such as tetanus [34] and botulism.[35] There have been many cases reported. They all follow the pattern of respiratory depression with coma and cerebrovascular collapse. The following case history of diphenhydramine poisoning illustrates the general pathological picture.

Case 4.—A 20-month-old girl in coma was admitted to the emergency department May 20, 1952, with a history of having ingested 1,200 to 1,300 mg. of diphenhydramine two hours previously. While the stomach was being gavaged, generalized convulsions developed and signs of vascular collapse were evident. The temperature was 104 F. Intravenous thiopental (Pentothal) sodium was given for the convulsive seizures. With definite signs of respiratory failure and the presence of continuous convulsive seizures, a tracheotomy was performed immediately. Ventilation improved, with recurrent bouts of respiratory depression still persisting. Continuous tracheotomy suction relieved this embarrassment. Coma persisted, and a generalized edema of the body and extremities occurred within three days. Tracheobronchial toilet was continued. A thick, tenacious, blood-tinged fluid was aspirated on the 20th postoperative day; the temperature rose to 104.3 F., and the respirations also rose to 68 per minute. Bronchopneumonia was diagnosed, and x-rays showed a collapse of the right upper and middle lobes. Atelectasis had occurred in spite of suction, and massive doses of antibiotics were then given. The patient died on June 13, which was 24 days after admission. The autopsy report revealed (1) cerebral edema with cortical neuronal degeneration, (2) poisoning attributed to diphenhydramine, and (3) bronchopneumonia of the right upper and middle lobes.

Comment.—Laryngeal and bronchiolar spasms, with early signs of respiratory failure, occurred simultaneously with the convulsive seizures, and tracheotomy was necessitated. Cerebral anoxia, with edema and hemorrhages, was most likely the result of respiratory failure combined with the toxic effect of the diphenhydramine. The clinical development of massive pulmonary collapse and subsequent pneumonia, in spite of adequate suction, was an interesting development. The generalized edema developed as a result of vascular collapse with renal shutdown, which was not reported in the autopsy findings. The die was cast with the overdose of diphenhydramine, and subsequent treatment was futile.

Acute Laryngotracheobronchitis.—Davison [29] and von Leden [38] have reported on acute laryngotracheobrochitis. This is a fulminating infection which affects both the upper and lower respiratory passages. Tracheotomy by-passes the laryngeal obstruction, and by suction through the tracheotomy the lower tree may be cleared. The following case report is submitted.

CASE 5.—On March 10, 1951, an infant was admitted to the hospital with a 14-hour history of crowing respiration, indrawing of the lower ribs, and a rectal temperature of 105 F. Endotracheal suction showed thick mucopus in the respiratory passages. Laryngoscopic examination showed marked edema of the epiglottis, and the vocal cords were difficult to visualize without traumatizing. The chest movements were markedly diminished. X-rays showed bilateral bronchopneumonia. Severe secretional anoxic signs were evident. An early tracheotomy was performed. This was done immediately after the findings at the time of endotracheal suction. Suction produced large amounts of mucopurulent exudate. Catheter oxygen was then given through the tracheotomy tube. Penicillin was also given. Within 12 hours the respiratory distress had subsided, and within 15 days the chest had cleared. The tracheotomy tube was removed at this time. The child made an uneventful recovery.

Comment.—The interference with alveolar ventilation was caused by atelectasis and bronchopneumonia as a result of bronchiolar obstruction [1] by mucopurulent exudate. The respiratory embarrassment subsided quickly, and the atelectasis and bronchopneumonia resolved satisfactorily as a result of the tracheotomy suction. Adequate antibiotic therapy and humidified oxygen are necessary aids to the tracheobronchial suction in the rapid resolution of the disease. A number of case studies reported no deaths when early tracheotomy was done.‖

Respiratory Obstruction Due to Foreign Body.—Sudden obstruction of the upper and lower respiratory passages by aspiration of foreign bodies is common. Any number of case studies stress the necessity for the early removal of these foreign bodies to restore the airway. They may be removed by the direct laryngoscopic route or by bronchoscopic procedure. Overwhelming secretions in the lower respiratory passages produce sudden asphyxia, and atelectasis may result. The following case report illustrates these points.

CASE 6.—On March 3, 1949, a 14-month-old girl aspirated food. The inhalator squad was unsuccessful in their attempt at resuscitation, and the child was admitted to the emergency department. Signs of respiratory obstruction with anoxia were present. Chest movements were diminished. Bronchoscopic suction on admission revealed a large quantity of milky vomitus, and signs of secretional anoxia did not entirely disappear with tracheobronchial aspiration. A tracheotomy was performed within 24 hours, and more efficient suction was made possible. Atelectasis and bronchopneumonia followed, which did not clear until the 21st day. The tracheotomy tube was then removed. Subsequent examination revealed mental retardation, which could be attributed to cerebral anoxia following the aspiration.

Comment.—Secretional anoxia persisted after the bronchoscopic suction, owing to the copious amount of foreign body present and the irritation of the bronchiolar membrane with edema and subsequent alveolar fluid accumulation. A tracheotomy was performed within 24 hours. The effects of the cerebral anoxia on the neuronal cells, however, were apparent at a later date, as evidenced by the mental retardation. Galloway repeatedly states that anoxic effects on the brain cell can occur within three to eight minutes. Therefore, a diagnostic bronchoscopy with an early tracheotomy was desirable.

‖ References 29 and 38.

Guillain-Barré Syndrome (Infectious Polyneuritis).—Difficulties may be encountered in differentiating some types of infections involving the central nervous system, such as the encephalitic manifestations of virus diseases, namely, measles and mumps. Other diseases which produce toxins, such as tetanus and botulism, may be included. The overwhelming effects of paralysis of the central nervous system causing respiratory embarrassment are indicated in the following case report.

CASE 7.—A 13-year-old girl made an uneventful recovery from measles until March 17, 1950, when she complained of headache, and pain and numbness in both hands and legs. A motor paralysis of the upper and lower limbs followed. Examination on admission revealed an interference with the cough reflex and swallowing mechanism. A bilateral facial paralysis was also noted. Anoxia was imminent. A spinal tap yielded fluid containing 300 mg. of protein per 100 cc., with no increase in cells. She was placed in a respirator and a tracheotomy performed. Suction revealed a large amount of mucinous exudate. Humidified oxygen was administered through a catheter. Levin-tube feedings were given. Chest complications were averted by tracheotomy with suction, and muscle tone appeared within two weeks of the surgical procedure. The tracheotomy tube was removed on the 26th postoperative day following the return of the cough reflex and of pharyngeal sensation. There was considerable wasting of the muscles of the extremities. Bilateral facial weakness persisted. Muscular tonus and power returned to the patient so that she was able to walk from the hospital unaccompanied.

Comment.—Considerable effort is sometimes necessary in differentiating this syndrome from bulbar and bulbospinal poliomyelitis. The spinal fluid findings and the early recovery of muscular tonus are helpful. Pulmonary complications follow a similar pattern, owing to the secretional anoxia, and treatment is exactly the same. An early tracheotomy is desirable in selected cases.

Head Injury and Barbiturate Poisoning.—The retention of bronchiolar secretions and consequent postoperative atelectasis were first described by Lee, Tucker, and Clerf.[1] The work of Atkins [31] and Arhelger [32] indicates the necessity of early tracheotomy in severe head injuries and in neurosurgical procedures about the head and neck to prevent pulmonary complications. A similar pathological pattern of pulmonary complications is seen in drug poisonings. The following case report is an example of a combination of a head injury and barbiturate poisoning.

CASE 8.—On Aug. 3, 1953, a 50-year-old man was found unconscious in bed, with an unknown history on admission. He showed no response to painful stimuli. The pupils were pinpoint and the limbs atonic. Irregular respiration and a weak pulse were apparent. The temperature was 102.6 F., and he was unable to swallow. A bilateral subdural exploration did not reveal any evidence of hematoma. An endotracheal tube was left in situ. Within a few hours respiratory distress became more evident and anoxic signs persisted. An immediate tracheotomy was done. A chest examination with x-rays revealed atelectasis of the right lower lobe. Tracheobronchial toilet was maintained. When the endotracheal tube was removed during the preparation of the tracheotomy, it was found to be markedly obstructed with thick, tenacious mucinous exudate and early crusts. The tracheotomy procedure was completed, and a large amount of mucinous exudate was aspirated from the tracheobronchial tree. Respiratory distress and anoxia disappeared almost immediately. Rales at the right base diminished within a few hours. The patient opened his eyes and responded to painful stimuli. Within 48 hours the coma had disappeared and the chest cleared. The tracheotomy tube was removed on the fourth postoperative day. He made an excellent recovery and was discharged on the eighth postoperative day. Subsequent history revealed that the patient had received a severe concussion from a fall after taking an overdose of barbiturates.

Comment.—A similar pulmonary pattern in many of these cases is revealed with bronchiolar obstruction and atelectasis in cases in which coma persists for 24 hours or more. The endotracheal tube itself may become obstructed, and constant observa-

tion to maintain a patent lumen is necessary. Removal of the obstructed tube, together with a tracheotomy and easy suction, produced a spectacular clearing of the obstructed airway and a quick resolution of the atelectasis. Again the loss of cough and swallowing mechanism is noted in coma.

Fat Embolism.—Patients with multiple injuries of the upper and lower respiratory tract—directly or indirectly causing an obstruction to the airway by edema, hemorrhage, or an overflow of secretions—are particularly prone to develop pulmonary complications. Head injuries and fractures of the cervical spine may be included in this category. Many excellent reports, inclusive of Echols'[33] good results, are encouraging. The following case of fat embolism indicates the dramatic results of an early tracheotomy procedure to remove the respiratory obstruction and prevent pulmonary complications.

CASE 9.—In August, 1950, a 22-year-old logger was admitted to the hospital with multiple compound fractures of the mandible and comminuted fractures of the right femur and left tibia. He went into coma within 24 hours of reduction of the compound fracture, with consequent loss of cough and swallowing reflexes. Evidence of respiratory obstruction was present. Examination revealed laryngeal obstruction, tetany, and convulsions. Cheyne-Stokes breathing was noted, along with a tracheal tug. The chest was full of rales, and well-developed anoxia was present. A diagnosis of fat embolism was made, with petechiae present in the skin of the thorax and fat cells present in the sputum. A tracheotomy was done, and suction revealed thick mucinous exudate. A general improvement in respiration occurred within a few hours. The patient's general condition improved, and he regained consciousness within 24 hours. During his long postoperative orthopedic recovery, tracheobronchial toilet was maintained without any evidence of pulmonary complications. He made a complete recovery and has returned to his normal occupation.

Comment.—The problem of respiratory embarrassment following surgical manipulation of fractures of the mandible, neck, or chest must include consideration of a prophylactic or therapeutic tracheotomy, particularly in the comatose patient. When in doubt, a diagnostic bronchoscopy can be done, except in severe cervical fractures.

Basilar Artery Thrombosis.—In certain acute neurological cases, inclusive of encephalomyelitis,[12] and cerebral vascular diseases producing hemorrhage or thrombosis with potential prolonged respiratory obstruction, a tracheotomy is indicated. The following case report exemplifies the effectiveness of a prophylactic tracheotomy.

CASE 10.—A man developed sudden quadriplegia on Aug. 26, 1952. He was seen on Aug. 28, 1952, regarding possible tracheotomy. At that time he was still able to swallow, and secretions were minimal. He did not appear to have any upper respiratory obstruction nor interference with the lower tracheobronchial tree.

On Sept. 1, his condition deteriorated rather suddenly, with increased secretions, inability to swallow, and some respiratory distress. Tracheotomy was done, after which it was possible to keep the tracheobronchial tree free of secretions. His recovery was slow, and over a period of six weeks his swallowing mechanism gradually returned to a more normal state. Decannulation was done on Oct. 21, and he was transferred to a Veterans Administration Hospital, where his further convalescence was uneventful.

The clinical diagnosis was basilar artery thrombosis.

Comment.—The prophylactic tracheotomy prevented pulmonary complications, and the patient made a satisfactory recovery. That he has returned to his full-time lecturing duties as a university professor attests to the prevention of cerebral anoxia in this case.

Acute Bulbospinal Poliomyelitis.—The prerequisite in the relief of secretional anoxia is adequate tracheobronchial suction. The following case illustrates this point.

CASE 11.—On Aug. 4, 1953, a seriously ill 7-year-old boy with bulbospinal poliomyelitis was in a respirator. This child had had a tracheotomy seven days previously. He was very lethargic and in a semiconscious state. He had been receiving repeated suction at regular intervals. While the tracheotomy tube was being changed, the respirator having been disconnected, the anesthetist provided artificial respiration. During the change, the oxygen machine failed. The tracheotomy tube was changed quickly and the respirator reconnected. During the interval when the machine had failed, anoxia became obvious, and the child became restless. In spite of humidified oxygen by catheter, even though the machine was again functioning and the respirator was active, the anoxia persisted. At this point, very thorough tracheobronchial suction revealed tenacious secretions from both bronchi. After this suctioning, the boy's color improved and restlessness disappeared. Ten days later, he was out of the respirator at short intervals and proceeded to a satisfactory recovery.

Comment.—The drama of bronchiolar obstruction and its effect on alveolar ventilation has been seen on many occasions. This particular instance reemphasizes the effectiveness of an adequate tracheobronchial toilet for the patient with acute bulbospinal disease in a respirator when adequate catheter oxygen therapy and effective artificial respiration (in a respirator) did not relieve the anoxia. One must act quickly in carrying out the necessary procedures to restore the airway.

SUMMARY

A knowledge of respiratory physiology and pathology is required to understand secretional anoxia. The physiology and pathology have been briefly reviewed. The loss of the protective reflexes should alert one to the potential danger of secretional anoxia. In this state, because of occlusion of air passages at any level, by excess secretions or other causes, adequate aeration of the blood in the pulmonary capillaries is impaired, and thus anoxia develops. The immediate and most important effects of this anoxia are felt on the cortical and then the medullary cells of the central nervous system. These effects occur very rapidly (three to eight minutes), and the changes in the neuronal cells may be permanent. It is obvious, therefore, that if a tracheotomy is necessary to relieve the respiratory obstruction, it should be done early. Atelectasis is the commonest sequel to tracheobronchial obstruction and in the majority of cases is preventable by adequate tracheobronchial suction.

However, a diagnosis of anoxia is difficult to make because clinical signs are unreliable. Present laboratory tests are complicated and not satisfactory. It would therefore seem essential to devise a rapid and reliable test for anoxia, that is, an estimation of carbon dioxide and oxygen tensions in the alveolar air and an estimation of arterial carbon dioxide and oxygen tensions. With the establishment of a diagnosis of anoxia in this way, the necessity for tracheotomy would be clearly indicated. In a clinical borderline case in which the need for a tracheotomy is doubtful, it should be remembered that complications from the tracheotomy per se are negligible, whereas the effects of anoxia endanger life.

CONCLUSIONS

Constant observation for the early signs of secretional anoxia should be maintained in all cases in which there is a potential danger of its development. The essential treatment of secretional anoxia involves the continuous clearance of excess secretions from the tracheobronchial tree where the normal protective mechanisms

have been damaged, as in the comatosed patient and in paralysis of the respiratory muscles. The most effective way of clearing the tracheobronchial tree is by means of a tracheotomy opening. The early use of tracheotomy in secretional anoxia is advocated as a means of prevention of the development of serious sequelae, such as atelectasis and severe anoxia.

Dr. Robert B. Kerr, Professor of Medicine, University of British Columbia, read the manuscript and gave his critical comments.

Dr. N. J. Blair granted me permission to use five of his case reports.

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REEVALUATION OF AIR-CONDITIONING FROM THE POINT OF VIEW OF OTORHINOLARYNGOLOGY

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AN EVER-EXPANDING sector of public and private life has been brought under the influence of air-conditioning. From a luxury and an exceptional attraction, air-conditioning has become a necessity of everyday life and constitutes in many climatic regions part of the standard indoor environment of modern man. All along, observations have been made on the physiologic effect of air-conditioning and its therapeutic applications. But engineering has taken the initiative, and there is a lag before medicine is able to catch up to the technological development.[21] By now, however, the time may have come to reevaluate the effects of air-conditioning on patients with acute or chronic diseases of the ear, nose, and throat. While atmospheric factors, including air-conditioned environments, affect the entire organism, their impact is probably most immediate with regard to the upper respiratory tract.

In the following survey it is taken for granted that the air-conditioning system is correctly designed, is expertly installed and maintained, and is serviced and checked at appropriate intervals by well-trained personnel. There is no saying what calamities might happen if even the best air-conditioning system is mismanaged or when mechanical breakdowns occur. Obviously, no medical evaluation of the effects of air-conditioning can concern itself with technical imperfections, although the possibility of such accidents cannot be completely ignored.

Air-conditioning of homes and public places is intended to establish an optimal degree of bodily comfort and at the same time to maintain normal performance and efficiency under adverse atmospheric circumstances.[11] In this respect, general air-conditioning differs essentially from specialized systems, designed for hospitals, particularly operating rooms, nurseries for premature infants, wards for infectious diseases, etc. Experience in such specialized settings can often contribute to improve the use of general air-conditioning for comfort but in itself is outside the scope of the present study.

Air-conditioning for comfort has been perfected to such a degree that it can now be "tailored to fit"[18] the specific requirements of various types of buildings and their respective occupants. Only a decade ago it was considered impracticable or impossible to design installations which would make more than 50 to 65% of an average group of workers, shoppers, or movie-goers comfortable at any given season,[28] but in the meantime it has become possible to provide indoor conditions which are satisfactory to 95% of the tenants.

The atmospheric factors determining human comfort are so well understood that there is no need to dwell upon them at great length. It is true, no objective index is available, and many variables cannot yet be quantitatively evaluated.[39] Comfort is

a complex situation which reflects the degree of strain imposed upon the heat-regulating system of the body in maintaining the balance between heat production and heat loss. Thermal comfort is determined by environmental factors like air temperature, relative air humidity, air movement, and radiation of surrounding structures; but it is also affected by clothing and physiologic conditions, particularly the state of health, sex, age, physical activity, and acclimatization. In addition, the sensation of comfort is influenced by psychic reactions. Thermal comfort thus depends on a wide range of components, among which only a limited number can be influenced by means of air-conditioning.

Control of indoor temperature is merely one aspect of air-conditioning, and measurements with a dry-bulb thermometer give little information as to the comfort or discomfort of the occupants. The rate of dissipation of heat from the human body at any given dry-bulb temperature depends largely on the water content of the surrounding air. The relative humidity of the environment is therefore of considerable importance for human comfort, and the readings of both temperature and humidity are combined by the use of the wet-bulb thermometer.[3] Air movement accelerates the exchange of heat and decidedly contributes to comfort in a humidity-saturated atmosphere. But it is difficult, if not impossible, to unify the values for air movement and radiation of surrounding structures into one single index. Effective temperature, for instance, combines air temperature, relative humidity, and air movement into one single value, which, however, would be meaningful only for the comfort of an average person, wearing normal clothing, while resting or remaining in a state of limited activity.[6] Yet, the comfort experienced depends, as mentioned before, on so many heterogeneous factors that estimates are preferably based on wet-bulb readings, making due allowance for other components.

In the evaluation of the influence of air-conditioning on the health or disease of the ear, nose, and throat, the air temperature as established by dry-bulb readings is of importance only in two respects. Insufficiently controlled indoor temperature causes steeply contrasting degrees of warmth at different room levels. Such a temperature gradient may, for instance, be present between the head and feet, resulting in chilling of the lower extremities while the head, especially the nose, feels stuffy and congested.[1] Subjects with a tendency to vasomotor disturbances are then apt to experience attacks of vasomotor rhinitis, and chilling of the feet can bring on a cold.

Still more pronounced temperature differences may exist between the controlled thermal environment indoors and the natural atmosphere outside. For the sensation experienced on entering from a hot outside atmosphere into an air-conditioned room of decidedly lower temperature the term cold shock is used.[8a] But the opposite change, coming from a cold space into the hot outside air, is similarly uncomfortable, and both experiences are described as air-conditioning shock.[22] The difference between the two temperature levels often amounts to 20 F., sometimes reaching an extreme of 30 F. Rapid transition from one temperature to another always imposes a strain on the heat-regulating mechanism of the human body, which, however, a healthy person will balance without difficulty; but subjects with a tendency toward hay fever or vasomotor rhinitis are less able to adjust quickly and show an increased susceptibility to infections, especially rhinitis and other upper respiratory involvements.[10] In experiments with guinea pigs the highest degree of sensitization and shock was observed when the animals were shifted from one temperature extreme to another.[9] The danger inherent in air-conditioning shock has long been recognized,

and some buildings provide for gradual adjustment by keeping hallways and lobbies at an intermediate temperature. Yet such installations are not always feasible, and it is necessary to advise patients to use appropriate clothing in order to mitigate the effect of air-conditioning shock.

Air-conditioning shock must especially be avoided when alighting from a train or airplane. With the extraordinary speed of modern transportation, the change from the protective air-conditioned environment to the outside atmosphere may coincide with a transition from one climatic region to another. Thus demands on physiologic adaptation become cumulative, and conditions connected with flying are apt to make sensitive persons still more responsive to sudden temperature changes. In such circumstances wryneck or an acute attack of sinusitis often follows.

In view of the hazard of air-conditioning shock, it is desirable to limit the difference between outside and inside dry-bulb temperatures to 5 degrees (F.) and to effect bodily comfort by changes in air humidity. But this is not feasible under all climatic conditions; for instance, with very hot and dry outside air, as it is encountered in deserts during the summer months, comfort demands an increase in indoor humidity combined with a more pronounced lowering of the dry-bulb temperature. Air-conditioning standards developed for summer cooling in the cold climates must be modified for the tropical and semitropical zones,[30] and basic research to this effect is under way at several universities.

The relative air humidity in air-conditioned environments is likely to exert an important influence on the upper respiratory tract. The human body is well adapted to compensate for wide variations of relative humidity, and evaluation of its effect on comfort and health must therefore remain approximate.[23] At present, an extreme range of relative humidity from 30 to 65% is assumed to be tolerable, with an optimum of between 45 and 50%. It is also recognized that summer air-conditioning usually calls for dehumidification, while in winter a higher humidity than that on the outside should be provided. Low relative humidity may predispose to respiratory diseases and incidentally also to nervous disorders. Patients with vasomotor rhinitis will do best in an air-conditioned room with a relative humidity of ±55% and a dry-bulb temperature of 70 to 72 F.[35] Subjects suffering from frontal sinusitis and other infections of the upper respiratory tract breathe more easily and experience greater comfort in a humid atmosphere as long as the acute attack lasts but will prefer a somewhat dryer environment after the condition has cleared up.[37] But not all observers agree on this point, some even reporting that a level of relative humidity as low as 35% is most favorable for patients with acute sinusitis and other respiratory infections.[26] Very low humidities (15% or less), however, are detrimental to the comfort of all subjects and result in drying of the nasal mucosa. As this latter effect is especially often observed during midwinter,[4] well-regulated air-conditioning might be expected to lower the incidence of respiratory infections toward the end of the cold season.

Some of the seeming contradictions with regard to the effect of various levels of air humidity on ear, nose, and throat diseases are obviously due to the general atmospheric conditions at the place of observation. In the wet and windy English climate the patient with hay fever or asthma obtains relief from reduced relative humidity.[26] But reports from New York City make it plain that hay fever, vasomotor rhinitis, chronic sinusitis, and similar complaints will be improved not only in a dryer atmosphere but also at coastal resorts where the air is as humid as in the city

but is continuously ventilated by mild breezes, not stagnant and stifling.[30] At high altitudes and in arid or semiarid regions, permanent residents are adjusted to a much lower level of air humidity than that tolerated by city-dwellers in wet climates.

Another factor in air-conditioning is control of air motion. For general comfort its rate must be adjusted to the prevailing temperature and therefore should be higher in summer than in winter. Persons subject to ear, nose, and throat conditions will benefit from the absence of drafts. An air velocity necessary to give the average occupant of an air-conditioned environment the sensation of freshness may be sufficiently strong to cause in sensitive persons reflex vasoconstriction of the skin and vasodilatation of the nasal mucosa, resulting in discomfort and occasionally even in upper respiratory disease. Whenever someone complains of defective air-conditioning, therefore, one should investigate whether the fault is not rather with his vasomotor response, heat regulation, and circulation.

Bodily comfort is also affected by the constant or oscillating character of air currents. Uniform air movement is registered as monotonous, while a limited degree of variability exerts a stimulating effect.[2] In natural circumstances the body is geared to transient rhythmic fluctuations, but a mechanical system gives off a constant flow with minimal variations and thus produces a feeling of stagnation.

The sensation of freshness, so ardently desired by patients complaining of stuffy noses, depends, in addition to relative humidity, air movement, and adequate exchange of air, on the radiation from surrounding surfaces. Atmospheric conditions which tend to reduce intranasal cooling increase vascular congestion, giving rise to sensations of stuffiness or even to actual obstruction of nasal breathing.[24] In tropical and subtropical regions it is desirable to keep the walls cool, as lack of heat absorption by the surrounding structures creates a feeling of oppression.[36] However, recent experiments have demonstrated that in a cooler climate walls warmer than the air in the room impart an impression of freshness.[24]

Thermal comfort depends, furthermore, on adjustment of air-conditioning in accordance with the location of the controlled environment. For instance, a room with a southern exposure requires less heating in winter, more cooling in summer. Similar differences result from the direction of prevailing winds, and high buildings must be divided into horizontal as well as vertical air-conditioning zones.[7]

Air sanitation is not the primary aim of air-conditioning, but it is often asserted that a controlled atmosphere provides a more healthful environment through reduction of the rate of air-borne bacteria and other pathogenic impurities. It has not yet been definitely established which role, if any, air-borne infection plays in the causation of upper respiratory diseases. For the purpose of an evaluation of air-conditioning, however, it will be presupposed that infection by the aerial route is of etiologic significance.

In crowded places we are breathing one another's respiratory flora,[5] and the question must be asked whether public air hygiene cannot be improved by means of air-conditioning. It has long been known that the load of carbon dioxide, other gases, and air-borne infective bodies shows a rising curve when all or part of the air of a conditioned space is recirculated.[10] In a recent series of experiments [16] it was demonstrated that office buildings and hotels which were not ventilated showed a total micro-organism count of more than eight times that of the outside air. Ventilation by dry coil systems, i. e., without washing, reduced the micro-organism level to 4.6 times the outside value; when the air was treated in freshly cleaned washers using

pure water, the micro-organism count was slightly higher than without any washing. Dirty washers using contaminated water increased the micro-organism count to 16 times that of the outside air. But when a chemical disinfectant was added, the organism content of the recirculated air dropped to 1.6 times that of the outside level and could be maintained at that rate. At least 95% of the specific air-borne pathogens (Staphylococcus aureus, Streptococcus pyogenes, and Corynebacterium diphtheriae) were destroyed when the air was exposed to the disinfectant in the water for 40 seconds. The content of molds was slightly lower (0.7 times) and that of hemolytic bacteria only somewhat higher (1.3 times) than in the outside air. Figures like these demonstrate the importance of chemical disinfection of recirculated air.

Technologic imperfections which otherwise often pass unnoticed will show up under the more stringent sanitary requirements of air-conditioned operating rooms. Thus in a recently reported case [25] postoperative infections were traced to highly contaminated soot which had by-passed a poorly fitted filter in the intake duct. But postoperative infections occurred again after this trouble had been corrected. They were then found to be due to a contaminated substance originating from reaction of chemicals in the city water on the metal of the discharge duct. Nothing can be taken for granted in planning, designing, installing, servicing, and running an air-conditioning system. Only after all technological requiremets have been met can there be a certainty that an air-conditioning system is not going to increase contamination of indoor air.

During epidemics of influenza and other respiratory diseases, the outside air contains many infective agents or viruses, as demonstrated by samples taken from hoods of automobiles, outside doors of Pullman cars, and diesel locomotives in transit, hotel roofs, etc.[27] Ventilation alone is not sufficient to guarantee hygienic conditions, and the average humidifier will not significantly reduce the amount of dusts, pollens, and bacteria in the circulating air. An air-conditioning system provided with air washers and using an effective germicide will lower the indoor pathogen count.[8] Ultraviolet irradiation of the air supply is less efficient than washing with a germicidal agent diluted in water. Even if merely a reduction of the bacterial concentration is desired, a rather complex set-up will be required to provide for precleaning of the air and an adequate duration of exposure to ultraviolet radiation.[29] Another disadvantage of ultraviolet light is that it is likely to create ozone, which in larger quantities exerts an intoxicating effect.[8] A more recent technique—using an electrostatic precipitator and cellulose filter paper—resulted in almost complete elimination of dust and a very significant reduction of air-borne bacteria (95 to 98%).[17]

The number of pathogenic bacteria found in the air is extremely small, even during an epidemic, and secondary dispersion of infective material, especially from clothing and floors—so-called indirect air-borne spread—seems to be of much greater importance.[34] Observations at a boarding school demonstrated that air-borne infection is much more closely related to the activity of the occupants than to crowding and is due mainly to liberation into the air of infected dust particles from clothing, towels, handkerchiefs, bedding, and furnishings.[14] In another study, beta hemolytic streptococci were isolated from more than half of the sweepings of floors in public buildings (schools, dormitories, theaters, and hotels); while the incidence showed seasonal variations, essentially the same sanitary problems were found to exist in these community foci as in hospitals.[31]

Not only bacteria but also viruses may be dispersed as dust-borne agents.[20] The natural death rate of bacteria in the dark is very slow; but bacteria in dust particles stirred up by moving persons or other means die off within a period of 10 days if exposed to daylight, low intensity ultraviolet radiation, or fluorescent lighting. Tungsten-filament lamps, on the other hand, have no bactericidal influence whatsoever.[18] Ultraviolet light has a deleterious effect on eyes and skin; yet screening, which would protect the occupants, reduces its bactericidal action. It has therefore been stated that a maximum of window space is desirable in public and industrial buildings.[5] The present architectural trend in structures of this type, however, is toward completely enclosed air-conditioned spaces, practically excluding penetration of daylight. Under these conditions air sanitation depends not only on air-conditioning but also on whether the building is provided with fluorescent lighting. In turn, the germicidal action may be greatly diminished if the fixtures are attached to a high ceiling.[5] Furthermore, atmospheric humidity is a factor, at least in the bactericidal action of ultraviolet radiation.[19]

Hygienic control of the atmosphere in air-conditioned spaces represents a very intricate task, and many engineering problems are still awaiting solution. It is up to the otorhinolaryngologists, working in close cooperation with physiologists and bacteriologists, to clarify the hygienic requirements of people assembled for one purpose or another in enclosed spaces. Undoubtedly, two sets of standards apply—the one at times of rampant epidemics, when even ordinarily resistant persons are apt to fall prey to pathogens, and the other for particularly sensitive patients with a known history of repeated upper respiratory diseases and allergies.

At present the point has probably not even been reached where purification of the atmosphere will reduce the danger of contagion during epidemics. A set of precautionary measures might be worked out, to be put into operation before an emergency arises. The spread of contagion shows a dynamic pattern, and a single case can initiate a chain of growing generations of infection.[32] All air-conditioning installations should be carefully inspected and brought up to perfect operating condition before the beginning of the cold season, so that they will work at the peak of their efficiency. Air hygiene is as much in the public interest as pure food, pure drugs, pure water, or smog control, and either by law or by the initiative of management safety standards should be formulated and put into practice.

Beyond this immediate objective there lies the prospect of providing at some future date in air-conditioned spaces a favorable atmospheric environment for patients with chronic upper respiratory diseases. Clarification of the specific requirements can proceed only step-by-step. It is up to the otorhinolaryngologist to study the conditions under which his patients are working and living and thus arrive at a better understanding of their reactions to the environment provided by air-conditioning. The time for generalizations has passed, and we are about to enter the case-finding stage. The future development of air-conditioning for hygiene must be based on evaluation of case histories and follow-up during convalescence, continued whenever possible after the patient has returned to his normal routine. On the strength of such factual material, coordinated with experimental findings, one may hope to determine with a greater degree of accuracy the influence which air-conditioning has on the patient with upper respiratory disease and finally to arrive at medical standards of air hygiene, for which the engineering profession has long been clamoring.[22]

As long as we are not entirely sure of the role of air-borne pathogens in the transmission of upper respiratory diseases, the still-existing imperfections of air hygiene should not be unduly stressed and the accent placed rather on establishing comfortable living and working conditions.[33] To be sure, air-conditioning is only one factor contributing to the complex sensation of physical comfort, but it is often the only one which can be favorably influenced. Moreover, in climates with high average annual wet-bulb temperatures, and in surroundings lacking natural air circulation, or overheated by lights, machinery, or crowds, air-conditioning becomes an absolute necessity.[15] The physical comfort thus obtained may reduce the strain on persons susceptible to chronic or recurrent upper respiratory diseases and allergies. While not of primary therapeutic significance, symptomatic relief may be achieved, and the temporary feeling of well-being will help to tide vasolabile persons over otherwise critical conditions. With the establishment of a more equitable situation, the patient is afforded a certain degree of biological rest,[12] especially desirable for elderly patients, in whom an intercurrent upper respiratory infection might otherwise aggravate a chronic ailment.

The attempt to bring climate to man [35] can, however, not be completely successful for every last person. It is difficult to satisfy the preferences of all occupants, especially in localities frequented by persons engaged in varying degrees of physical activities. Yet, "with regard to artificial climates we somewhat resentfully expect that this air-conditioning fully will be adjusted to our requirements." [22] This error should particularly be pointed out to meteorosensitive persons, who will have to learn to adjust themselves as much to artificial as to outdoor weather. It is this very same group of patients in whom the differences between outdoor and air-conditioned environment is liable to upset the natural equilibrium, and the physician will have to decide how air-conditioning shock can be avoided. A few simple rules, repeated by the otorhinolaryngologist until the patient can no longer escape their impact, may promote a new protective behavior pattern. In order to guide the patient through these and similar difficulties it is desirable that the otorhinolaryngologist have a fair understanding of air-conditioning and its effect on patients with diseases of the upper respiratory tract .

SUMMARY

Air-conditioning for comfort depends on control of temperature, humidity, air movement, and radiation of surrounding structures.

The impact of each one of these factors on subjects with chronic or recurrent upper respiratory disease is discussed.

Air-conditioning shock due to transition from an outdoor to an air-conditioned environment, or vice versa, must be avoided or at least mitigated.

Air sanitation, especially control of the pathogen content of indoor air, is reviewed.

Otorhinolaryngologic observations may furnish standards for further technologic improvement of air-conditioning.

Meteorosensitive patients depend for optimal adjustment to air-conditioning on the advice of a well-informed otorhinolaryngologist.

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LOCAL USE OF HYDROCORTISONE ACETATE IN THE NOSE

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THE VALUE of the cortisone and cortisone-like (hydrocortisone) hormones in relieving symptoms and temporarily halting the altered processes of allergy has been adequately established and is well known.*

However, the problems incidental to their utilization on a systemic basis are considerable, and it is generally accepted that their systemic use should be limited to the temporary alleviation of severe reactions that endanger the vital functions of the patient.[2] It is obvious that few of the nasal afflictions fit into this category. Yet the problems incidental to nasal allergy are manifold and are difficult to control.† Nasal obstruction, nasal polyposis, postnasal drainage, chronic pansinusitis, catarrhal otitis media, and chronic bronchitis are closely related to the problem of upper respiratory allergy. Even though great advances in the treatment of these conditions have recently been made, especially by Hansel and his group, the management is not entirely adequate. It is apparent that investigators in this field must be on the alert for possible developments in further enhancing the management of these conditions.

The successful application of the corticoids locally in the eye,‡ skin,§ and joint surfaces ‖ stimulated thinking along this line in rhinological problems. The nasal mucosa is accessible and absorptive and so should react as favorably as other tissues to local applications of the hormone.

Recent publications have indicated that hydrocortisone (Compound F) is, as far as is presently known, the most important natural antiphlogistic corticoid.¶ Increasing reports indicate that it is an effective agent in suppressing hypersensitivity phenomena, with similar effects as cortisone but far more intense.# It likewise appears that hydrocortisone has greater activity at a local tissue level.* Thus, its local use in the nose in allergy offers intriguing possibilities.

Prior to the reawakening to the potentiality of hydrocortisone, cortisone (Compound E) had been used locally in the upper respiratory tract by a number of

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Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

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References 39 and 55.

* References 2, 23, 32, and 39.

clinicians, with rather indifferent success. The various means of applying the material in itself suggests that there was no one satisfactory method. Bordley,[42] as early as 1949, reported on the trial use of cortisone nose drops for relief of nasal allergy, with indifferent results. Dill [4] utilized a nasal spray of this hormone, with one-half of his small series showing temporary marked improvement. Gelfand [44] used aerosol cortisone in bronchial asthma and noted worth-while relief in four out of five patients. However, it is to be noted that 50 mg. of the hormone, a sizable oral dose, was given daily for two weeks. In a small group of eight patients, London and Alexander [10] reported considerable relief from the local instillation of cortisone suspension directly into the bronchial tree. Doerner,[46] however, in reporting on the use of an aerosolized suspension of cortisone, noted poor response. Wall and Shure,[7] in a very excellent preliminary report in August of 1952, suggest a favorable response in true nasal allergy, with poor response in the so-called vasomotor rhinitis. Their work entailed the injection of 2.5 to 25 mg. of cortisone into the submucosa of each nasal space.

After a review of the results of the various investigators, it must be concluded that although certain of the reports are somewhat promising, there are at present no definite criteria as to the usefulness of cortisone, locally or topically, in the treatment of respiratory allergy.

However, there has been a recent general realization that hydrocortisone, while similar to cortisone qualitatively in its major metabolic and therapeutic actions, possesses quantitatively a greater metabolic effect and has strikingly great effects of local administration.† It has also been shown recently ‡ that hydrocortisone has the same but a more intense effect in suppressing certain hypersensitivity phenomena.§

Clinical evidence is accumulating rapidly as to its superior action as a local application in the eye, in and on the skin, and in joint surfaces.

Goldman and Preston [25] in a considerable series of patients studied the local therapeutic effect of hydrocortisone in diseases of the skin. In their excellent and complete studies, they attempted to evaluate injections into the area, applications by hypospray, topical suspensions, ointments, and various solubles. In their hands, ointments were found to be generally disappointing except in a few cases. The direct injection of the suspension into the skin lesions showed the best results, with inhibition appearing in three days and persisting for some time. This report is of special interest in that it, together with earlier publications by this group of investigators,‖ shows an increasing enthusiasm for the local use of hydrocortisone in skin diseases. Sullivan [24] confirmed this in his study of the skin lesions of sarcoidosis. In his small but significant series all cases showed gradual complete or nearly complete local resolution, with no gross inflammatory reaction. In three of the five patients, recurrence appeared in four to seven weeks, but the recurrence was one-third smaller than the original. In the remaining two patients, there was no recurrence in 4 to 14 weeks.

† References 2, 23, 32, and 39.

‡ References 32 and 48.

§ References 32 and 48.

‖ References 26 and 32. Goldman, L., and Baskett, J.: Compound F in Dermatology, read before the Academy of Dermatology and Syphilology, College of Medicine, University of Cincinnati, Chicago, December, 1952.

Sulzberger,¶ likewise, in a substantial number of skin problems, found hydrocortisone more active and more effective in its local action than cortisone. He found the ointment to be effective in the majority of cases of atopic dermatitis. Further, he stated that in no case was there any clinical evidence whatsoever of adverse systemic effects produced by topical application of the drug, and there were no instances of allergic contact sensitization.

These reports, especially that of Sullivan, would seem to be quite significant. Definite localized improvement in the majority of cases was obtained for a considerable period of time when the hormone was successfully applied to the reacting tissue.

The usefulness of cortisone in the treatment of certain diseases of the conjunctiva, sclera, cornea, and anterior segment of the uveal tract was established by Steffensen [47] in 1950. This was confirmed, clinically and experimentally, by Duke-Elder,[20] Smith,[23] Steffensen, Ashton,[22] and others in 1951. However, in 1952 Steffensen # and his group adequately demonstrated the similar but increased effectiveness of topically applied hydrocortisone in anterior segment eye disease. In addition to the increased effectiveness of hydrocortisone, they noted that prolonged treatment with hydrocortisone did not seem to induce resistance to the substance, as was the case with cortisone.

Hydrocortisone has also been used successfully as a local antirheumatic agent by direct injection into joint cavities. Since the published results of Hollander,* Duff,[48] and others in 1951 on the intra-articular use of cortisone and hydrocortisone, much evidence † has appeared to show that hydrocortisone is strikingly superior in its effectiveness. While this action has been primarily antiphlogistic in nature (osteoarthritis, traumatic arthritis, gouty arthritis, acute bursitis), it demonstrated this substance's superior local activity.

GENERAL CONSIDERATIONS

It is necessary in the evaluation of allergic therapy not only to note apparent clinical responses but also to study all present knowledge and theorize the influences and actions of the agents used in an attempt to explain the nature of allergic response and offer a rationale of treatment.

The exact nature of allergy is unknown. However, Selye,[71] in his treatise based on the concept of the general adaptation syndrome, describes derailments of the adaptive mechanisms as the principal factors in the production of certain maladies. One of these is the response of a shock organ to an allergen. He stated that the response of the adrenal cortex is most important, not only in defense against systemic stress but also in the manifold topical defense reactions which occur upon exposure to local stress (e. g., responses of a shock organ to an allergen).

In this response of the shock organ to an allergen, certain facts have been observed. It is known that in the shock organ fluid escapes into the ground substance of the tissue, with changes in both the ground substance [49] and in the connective tissue cells.[51] The mechanism of this escape of fluid is not clearly understood. Rawlins [54] cites the work of certain investigators which indicates that this edema

¶ References 28 and 29.

References 18 and 19.

* References 35 and 39.

† References 31, 35, 36, and 38.

may be due to liquefaction of the ground substance about the capillaries, thus loosening the endothelial connections from without.[50] However, in the shock organ response to an allergen, there also appears to be some increased capillary permeability due to some direct action on the intracellular cement of the capillary endothelium. It is also known that in connective tissue damage from any stress, including allergy, there is an increased liquefaction or depolymerization of the ground substance, i. e., the mucopolysaccharides are broken down into their small aggregates.[52] This breakdown is characterized by increased fluidity of the ground substance, increase in ground substance, and seepage of ground substance into the circulation.

The knowledge that hyaluronidase [50] can do this and that there is a measurable increase in nonspecific antihyaluronidase in the blood in connective tissue damage due to stress ‡ would suggest that hyaluronidase plays a major, yet ill-defined, part in this picture. It would seem safe to accept Rawlins' [34] view that this system is in some manner connected with the stress hormones of the adrenal cortex.

Much inferential evidence has been accumulated to indicate that certain adrenocortical extracts have a counteracting effect on the increase in capillary permeability produced by many noxious agents.[54] However, it is not known whether this action is the result of distribution of blood to a given area or is due to the direct action of the hormones in maintaining the structural integrity of the capillary walls or to some indirect action on the ground substance.

Clinically, it has been repeatedly observed § that corticotropin (ACTH) and cortisone reverse the changes seen in allergic phenomena, with a restoration towards normal in all phases of ground substance and cellular activity. It has been suggested that these hormones exert their action through some alteration of the antibody-antigen system.[8] However, this has been disproved by recent studies of Cooke,[13] Feinberg,[14] and others.[45] From these reports, it would appear that the observed effect of these hormones in altering the sensitivity or immune reactions, at least in the human, is entirely nonspecific and is not intimately related to the antibody-antigen mechanism.

The exact part played by the release of histamine or histamine-like substances by damaged cells is likewise not clear. However, it is known that these substances do stimulate the parasympathetic system to various grades of activity, with acetylcholine entering the picture. This apparently throws the adrenogenic-cholinergenic system out of balance, with increased cholinergenic activity. The effect of this is also increased capillary permeability, with leakage of fluid into the surrounding tissue. Although this process theoretically takes place in clinical allergy, the exact mechanism of this action cannot be clearly explained. It has been adequately demonstrated that intense adrenocortical stimulation does not significantly alter the ability of tissue to liberate histamine [59] or histamine-like substances.[60] It likewise does not alter the tissues' ability to respond to these substances.|| Yet the ability of cortisone, hydrocortisone, and the adrenocorticotropic hormones to control this phase in allergic reactions is a known clinical fact. It would appear that the poorly understood action of these hormones must be on the ground substance of tissue.

‡ References 40 and 53.

§ References 3 and 55.

|| References 59 and 60.

It has been adequately demonstrated that certain of the pituitary and adrenal hormones, mainly cortisone and hydrocortisone, termed the antiphlogistic hormones by Selye, depress the production of all elements of connective tissue and decrease the permeability of the ground substance. Apparently, antagonistic to these are Selye's prophlogistic adrenal and pituitary hormones (such as desoxycorticosterone and somatropin), which increase the ground substance permeability. Other hormone systems also enter into this in a lesser degree. The thyrotropic hormone effect in stimulating ground substance and collagen is well demonstrated in clinical exophthalmos. Likewise, the ability of the estrogenic hormones to decrease ground substance permeability and the antagonistic effect of the gonadotropic hormones have been well established.[61] However, it appears that the adrenocortical hormones are the principal agents associated with this activity.

It has also been established that local application of the antiphlogistic corticoids lessens an inflammatory process, indicating that this action is at a local tissue level. Selye, in his pneumoderma experiments, noted that hydrocortisone, which is, as far as is now known, the most active natural antiphlogistic corticoid, had prolonged effects on tissue. He postulated that these effects were due to either prolonged hormonal fixation in tissue or possibly sensitization of the tissue to the anticonnective tissue and antiphlogistic actions of hydrocortisone. If the former is true, the topical action of hydrocortisone is greatly augmented. Selye found that the effect of 1 mg. of hydrocortisone in tissue was undiminished after two months.

From the foregoing data, it would seem safe to theorize that the observed favorable effects of hydrocortisone, cortisone, and corticotropin on the allergic processes lay in the alteration of the stress reaction of the shock tissues. In this regard, it is further theorized that this alteration is invariably due to a nonspecific inhibition of the response in the target area and is probably not related to actual interference with antibody formation or with the union of antibody and antigen. This response inhibition is most likely mainly the result of the influence of this hormone group on the hyaluronidase system together with some direct action on capillary permeability. There is probably little or no direct hormonal effect on the associated parasympathetic stimulation. It does not appear that the local formation of histamine and histamine-like substances is reduced by the action of these hormones,[60] but somehow the tissue reaction to these substances is reduced. The mechanism of this action has not been satisfactorily explained.

From the foregoing data, it would seem safe to postulate that adequate topical or local application of these hormones to the shock organ in nasal allergy (the nasal mucosa) would successfully hinder or alter the stress reaction of the tissue. In that hydrocortisone appears to be the most active known antiphlogistic and antiallergic corticoid, this substance theoretically should be an effective topical or local agent for suppressing allergic-type reactions if it could be successfully applied to the involved tissue. This would be of great clinical importance, as it would produce the desirable local effects without the unwanted systemic effects of corticoid therapy. If hydrocortisone could successfully be applied and the action be as expected, its usefulness in the care of nasal allergy would be considerable.

However, it is not considered that this type of therapy will in any way be a substitute for the accepted hyposensitization treatment process but rather act as an adjunct in alleviating the tissue reaction until hyposensitization has successfully controlled the antigen-antibody reaction.

ADRENOCORTICAL PHYSIOLOGY

The adrenocorticotropic hormone corticotropin is secreted by the anterior pituitary gland, apparently mainly in response to stimulation from the hypothalamus. This, in turn, is stimulated by epinephrine (a product of the adrenal medulla) and probably directly by the central nervous system.

Corticotropin, in turn, stimulates the adrenal cortex to secrete many biologically active steroid hormones. As yet, there is no full understanding of the multiple activities of these hormones, just as there is no definite knowledge of the total number secreted. Approximately 28 different steroids have been identified, but of these only 6 have been shown to be biologically active. However, it is evident from their diverse and biologically important known activities that these substances are fundamental in one's life. Their relationship to stress and the reaction of the organism to stress are apparent in the multiple nature of their effects on the body. Included in the known effects are control over electrolyte and water metabolism, control over the metabolism of carbohydrate, protein, and fat, some control over sex function, and control over tissue reaction to stress. The activity of these hormones produced during stress depends on a variety of conditioning factors.[62] These include heredity, age, previous exposure to stress, nutritional state, etc.

In the consideration of allergic phenomena, the actions of certain of these corticosteroids are of prime importance. Although the influence of hydrocortisone-like substances on the metabolism of the constituents of connective tissue is only partially understood, they are known to inhibit the synthesis of chondroitin sulphate, a constituent of connective tissue ground substance.[63] They are thought to increase the degree of polymerization of hyaluronic acid of joint fluid in rheumatoid arthritis.[43] They are known to inhibit the activity of hyaluronidase in vivo.[64] In electrolyte metabolism, they cause retention of sodium and chlorides and increase the excretion of potassium. This results in an intracellular depletion of potassium, with its replacement with sodium. Likewise, there is a decrease in intracellular water content, with an increased intercellular content without a marked change in total fluid volume.[65] It is also known that these substances stimulate protein catabolism, with an increase in urinary excretion of histidine, glutamine, alanine, glycine, and lipin. In addition, the hydrocortisone-like substances produce neutrophilia,[66] lymphopenia,[69] and eosinopenia.¶ The latter two are apparently due to direct action on the cells, with an antagonistic effect by heparin.# These hormones are also known to have a depressing effect on the basic processes of inflammation, with decreased vascular permeability, diminished exudation, impaired migration of inflammatory cells, and depression of all components of connective tissue. This results in a decrease of fibroplasia and a shrinkage of collagenous fibers.

From the preceding summary, it is evident that knowledge of the actions of these glucosteroids is fragmentary and not clearly understood. It is also apparent that in our present state of knowledge the systemic administration of these agents is not to be taken lightly, and their use should be reserved only for temporary states in which vital function of the organism is in danger. The marked activity of

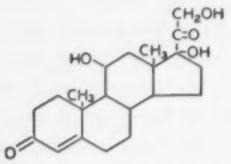
¶ References 67 and 68.

References 67 and 70.

these substances at the local tissue level would indicate the need of further investigation into methods of application locally and in this manner avoid the unwanted and dangerous systemic reactions to their systemic use.

DESCRIPTION OF HYDROCORTISONE

Hydrocortisone (17-hydroxycorticosterone), Kendall's Compound F, differs from cortisone in only a single structural detail—substitution of a hydroxyl radical for a ketone group of the C-11 position in the steroid nucleus.



Hydrocortisone. Cortisone differs only in that the OH group attached by a single bond at C-H is replaced by O attached by a double bond.

Hydrocortisone is available in two forms—the pure hormone (free alcohol) as it is extracted from the adrenocortical tissue and the partially synthetically prepared hydrocortisone acetate. The free alcohol form is difficult to extract and at present is available only in small quantities. Significant differences in potency are present between the two forms, with the hydrocortisone acetate being approximately one-half as potent, milligram for milligram, as the hydrocortisone (free alcohol). Hydrocortisone (free alcohol) is far more soluble in various media, including tissue fluid, than is hydrocortisone acetate. This solubility difference may prove to be of marked therapeutic significance.

TOXICITY OF HYDROCORTISONE

Hydrocortisone systemically produces the same general effects as cortisone. A recent publication by Salassa and his associates [33] points out the serious depressing effects of these substances on the adrenal cortex itself, with the possibility of an acute adrenal cortical insufficiency in the face of slight additional stress.

However, in the small doses used topically or locally, this problem has been obviated. It would seem highly improbable that any reaction would be encountered in this form of therapy. The possibility of an allergic reaction to the substance itself must be considered. (This apparently occurred in one patient in my series.) No other reactions were seen.

PREPARATIONS AVAILABLE FOR STUDY

The preparations * used in my experiment were the following: a suspension of hydrocortisone acetate (25 mg. to 1 cc. suspension agent), nasal jelly of 2.5% hydrocortisone acetate, and nasal jelly of 2.5% hydrocortisone and 5% pyrilamine maleate.

* Dr. Frederick K. Heath, Merck & Company, Inc., Rahway, N. J., provided the material used in this study.

Unfortunately, hydrocortisone (free alcohol) was not available in sufficient quantities for this study.†

METHODS OF ADMINISTRATION

Suspension of Hydrocortisone Acetate.—The suspension of hydrocortisone was injected by a long thin needle into the submucosa of the inferior and middle turbinates. An attempt was made to inject small quantities into multiple areas; 0.5 cc. of the suspension (12.5 mg.) was injected into each side. At first, this was done one side at a time, but, as no undesirable reactions were observed, both sides were injected at one time in the later cases.

Nasal Jelly of 2.5% Hydrocortisone Acetate.—In all patients the material was carefully spread over the visible and available mucosa of the nose. Certain patients were given the material to use at home with definite instructions as to methods and frequency of use. The objective and subjective results were carefully tabulated. The accepted methods of the treatment of nasal allergy were carried out in some cases simultaneously and in others at a later date. This will be commented upon later. In the cases of nasal allergy with nonsuppurating maxillary sinusitis, the jelly was instilled into the sinus through a van Alyea natural orifice cannula.

Nasal Jelly of 2.5% Hydrocortisone Acetate and 5% Pyrilamine Maleate.—This material was applied in the same manner as the plain 2.5% hydrocortisone acetate jelly.

SELECTION OF PATIENTS

A total of 53 patients with proved upper respiratory inhalant allergy were selected from my private practice. In this group were included patients with both the seasonal and perennial types. Ragweed pollen, various grass pollens, certain mold spores, and house dust were the principal offending allergens. The patients were classified into groups according to the severity of nasal symptoms and those with associated active paranasal sinusitis. The patients in the nasal group were divided into those with severe, moderate, and mild symptoms. Only five patients with associated chronic maxillary sinusitis were studied separately, but it was believed that this small number of cases should be included, as these seemed to follow a definite pattern.

The ages of the patients varied from 6 to 68 years. There were 31 males and 21 females in the group. All were otherwise in reasonably good health. Ten of this total group had associated asthmatic symptoms.

It is to be noted that other forms of therapy were not withheld in the severe cases. Hyposensitization was carried out within a few days and ultimately gave marked relief in the great majority. In fact, I gained a definite impression that following application of the hydrocortisone, hyposensitization seemed much more effective.

COMMENT

It is noted in the Table that the local application of 2.5% hydrocortisone acetate nasal jelly gave effective relief, both symptomatically and objectively, in four out of five patients who had mild symptoms. However, in the group with severe symptoms, only two had marked relief, with four having some relief and four having no improve-

† Dr. Frederick K. Heath revealed (after my thesis was submitted for approval) that the free alcohol of hydrocortisone is now synthesized and is commercially available as Hydrocortone tablets.

ment. In comparison, the local application of the nasal jelly containing 2.5% hydrocortisone acetate and 5% pyrilamine maleate seemed definitely less effective. Only 1 patient of this group of 15 had effective relief, although 12 had slight to moderate relief. The irritating qualities of the pyrilamine maleate were believed to account for the less effectiveness of this product. Mucosal reaction was increased in the majority, producing an unpleasant irritative sensation for several hours after application. The plain 2.5% hydrocortisone acetate suspended in the same nasal jelly generally relieved the itching and irritation already present and caused no subjective complaints. There was one exception to this in which a moderately severe local reaction occurred to the application of the plain 2.5% hydrocortisone jelly. This gave the clinical appearance of increased allergic phenomena, with complete nasal blockage and profuse rhinorrhea for approximately 24 hours. This patient refused further applications and so was classified as having no relief. There were no other reactions noted, either locally or systemically.

Results of Treatment with Hydrocortisone Acetate in Patients with Upper Respiratory Inhalant Allergies

Therapy, Type	Nasal Symptoms, Degree	Patients, No.	Definite Relief	Slight Relief	No Relief	Reaction
Local application of 2.5% hydrocortisone acetate suspended in a nasal jelly	Moderate severity Mild severity	10 5	2 4	4 1	4 0	Local allergic-type reaction (1)
Local application of 2.5% hydrocortisone and 5% pyrilamine maleate suspended in a nasal jelly	Moderate severity Mild severity	9 6	0 1	7 5	2 0	Unpleasant irritation noted for several hours by majority
Submucosal injection of 0.5 cc. hydrocortisone suspension submucosally into turbinates bilaterally	Marked severity Moderate severity Mild severity	8 4 3	2 0 0	4 3 2	2 1 1	None
Combination of submucosal injection of 0.5 cc. hydrocortisone suspension bilaterally followed by local application of 2.5% hydrocortisone acetate nasal jelly	Marked severity	3	1	2	0	None
Instillation of 2.5% hydrocortisone acetate nasal jelly into maxillary sinus	Moderate severity with associated maxillary sinusitis	3	0	0	3	None
Instillation of 2.5% hydrocortisone acetate with 5% pyrilamine maleate nasal jelly into maxillary sinus	Moderate severity with associated maxillary sinusitis	2	0	0	2	Severe headaches for 12 hours

Topical applications were not attempted in the severely reacting nasal allergies because of the obvious technical difficulty of applying a jelly to a completely blocked nose. There was a total of 11 such patients, in whom 0.5 cc. of hydrocortisone suspension (12.5 mg.) was injected submucosally into the inferior and middle turbinates of each nostril. The results were disappointing. This possibly was because of the inadequacy of the method rather than lack of activity of the hormone. The technical difficulty of spreading the material over a fair proportion of the reacting mucosa is apparent. Of this group of 11 patients, only 2 had effective relief, while 6 had some improvement, and 3 had none. In three of these patients, 2.5% hydrocortisone jelly was also used locally, but of this group only one of the three had marked relief, with the remaining two having partial relief.

Submucosal injections of the hydrocortisone suspension were also used in four patients with moderately severe nasal allergy and in three patients with mild nasal allergy, to determine whether the milder reacting mucosa would respond better. This seemed to be true with topical application of the jelly suspension. However, here the results were approximately the same as the severely reacting group.

The instillation of 1 cc. of 2.5% hydrocortisone nasal jelly into certain nonsuppurating maxillary sinusitis associated with perennial nasal allergy was dis-

appointing, with only slight relief in one case out of four. When 5% pyrilamine maleate was added to this suspension, there was the additional complaint of a secondary headache lasting for several hours. No improvement in symptomatology was noted.

It is also to be commented upon that in the group with asthma the local effects of hydrocortisone suspension controlled the nasal mucosal reaction in four cases and improved nasal symptoms in two additional cases but did not affect the asthmatic symptoms in any case. All of these were later satisfactorily controlled by hyposensitization.

SUMMARY AND CONCLUSIONS

The local application of a 2.5% suspension of hydrocortisone acetate in nasal jelly is of value in the treatment of nasal allergies. Its usefulness is apparently greatest in the milder, less reactive group. Specific hyposensitization, as outlined by Hansel, should be carried out in conjunction with the local application of the hormone, as there appears to be some unexplained synergistic effect that increases the effectiveness of both.

The injection of the suspension of hydrocortisone acetate (25 mg. per 1 cc.) into the submucosa of the turbinates proved to be of little value. This was probably the result of ineffective distribution of the hormone in the shock organ rather than due to inactivity of the hormone.

The addition of 5% pyrilamine maleate to the 2.5% hydrocortisone acetate nasal jelly seemed to decrease its effectiveness. This was believed to be due to the local irritant properties of the substance.

The intrasinus instillation of 2.5% hydrocortisone acetate suspended in nasal jelly in nasal allergy with nonsuppurating maxillary sinusitis appeared to be of little or no value. However, no effects or disagreeable symptoms arose as a result of this type of therapy.

There are definite indications that when hydrocortisone (free alcohol) or other, more soluble similarly acting hormones are available, the local use of these agents will improve the management of nasal allergy to a remarkable degree.

There was no clinical evidence of systemic reaction to this local hormonal therapy in any observed case.

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OLFACTORY ESTHESIONEUROEPITHELIOMAS OF LOUIS BERGER

L. FRÜHLING
AND
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THE PARTICULAR origin of the olfactory mucosa is well-known. Both the epithelial and the nervous portions originate from the same thickening, which forms above the rudimentary mouth to the detriment of the epiblast. It is therefore a rare, but no less unquestionable, example of the epiblastic origin of certain nerve cells.

One might expect, a priori, to encounter in the nasal fossae tumors bearing a more or less marked resemblance to those originating in the central nervous system. This fact has been definitely established. It is to Louis Berger that credit is due for the identification of the nerve tumors of the olfactory organ.

Berger [1] described in 1924, at the Institut d'Anatomie Pathologique de Strasbourg, a tumor of the nasal fossae, observed in a 50-year-old man, of which the histological structure was reminiscent of that of the embryonic nerve tumors, called "nevraxo-epitheliomas" by P. Masson and characterized by the presence of true cavities, the "neuroepithelial rosettes."

Following the appearance of this original case, Berger [2] published a second report in 1926 on a tumor of the nasal fossae, with a structure appreciably different from that of the first case. Indeed, the neuroepithelial rosettes were completely absent, and the tumor resembled a sympathoma fairly closely.

Certain of the tumors described below are olfactory esthesioneuroepitheliomas, but the majority have gone beyond the stage of the neuroepithelium and generally no longer display any neuroepithelial cavity. They have, on the contrary, undergone an almost exclusively neurospongial or sympathetic evolution. The cases are listed as follows:

One case observed by Portmann, Bonnard, and Moreau [3]; two by Escat [4]; one by Massier and Duguet [5]; four by Tavares [6]; one by Grigouroff and Dulac [7]; one by Wild, Frühling, and Klotz [8]; one by Martin, Dargent, and Gignoux [9]; one by Stout *; one by Lenz †; one by Seamon [10]; one by Huet [11]; one by Rossert and Chesseboeuf [12]; one by Terracol, Guerrier, and Barjon,[13] and one by Bosc and Browaeys.[14]

Satisfactory silver-salt techniques permitted the demonstration in 1936 by Wohlwill [15] of the neuroblastic potential of a true esthesioneuroepithelioma (i. e.,

From the Institut d'Anatomie Pathologique (Director, Professor Gery) and the Clinique Oto-Rhino-Laryngologique (Acting Director, Dr. Wild), of the Faculty of Medicine of Strasbourg.

* Cited by Seaman.[10]

† Cited by Seaman.[10]

one displaying the neuro epithelial rosettes), by providing evidence of typical unipolar and bipolar neuroblasts. This fact has recently been confirmed (in 1946) in a further true esthesioneuroepithelioma, by Ribeiro.[16]

The utilization, intensive or variable according to the seat of the tumor, of the multiform potential of the tumor matrix of olfactory neuroepithelium is sufficient explanation of the structural complexity, the polymorphism of the tumors, and the consequent diversity of the terminology employed by the various authors.

Finally, let us cite from among the recent general reviews concerning this question the work of Portmann and Beillard [17] and that of Terracol.[18]

It is obviously impossible, from the pathogenic point of view, to affirm that all nerve tumors of the nasal fossae originate in the embryonic neuroepithelium. It is probable that a certain number of tumors have their origin in dysembryoplastic neuraxial inclusions of the roof of the nasal fossae, in the sympathetic nerve fibers, or even in the sphenopalatine ganglion.

PERSONALLY OBSERVED CASES

We have noted in the records of the Institut d'Anatomie Pathologique de Strasbourg a total of four authentic cases of olfactory esthesioneuroepithelioma, examined between 1930 and 1952, of which we report the anatomic and clinical observations.

CASE 1.[8]—*Clinical Observations.*—Alfred K., aged 27, was sent to us on October, 21, 1947, by his doctor, for a slight blood-tinged mucous discharge from the left nasal fossa and an obstruction of the same.

The patient's previous medical history showed rachitis in early childhood, pneumonia at 9 years of age, temporal head pains and frequent pains in the nape of the neck since childhood which had tended to diminish during the last few years, exanthematous typhus during a 13-month period (1944-1945) as a deportee in the Dachau concentration camp, and from February to June, 1946, treatment in a sanatorium for suspected tuberculosis, a diagnosis not since confirmed. With the exception of tonsilitis, in 1933, there was no previous history of ear, nose, or throat disease. The patient had a marked tobacco addiction. The patient did not admit alcoholism or venereal disease.

The onset of the present illness went back to the first days of October, 1947; the symptoms were a slight obstruction of the left nasal fossa and burning nasal pains radiating towards the left eye. There was no development of this condition until the morning of Oct. 21, when, on blowing his nose, the patient noticed a slight blood-tinged mucus discharge (no pus). The left root of the nose was a little swollen and was painful when pressed. The patient's doctor on consultation immediately sent the patient to us for examination of the ears, nose, and throat and treatment.

Examination of the face revealed a very slight swelling of the left root of the nose, which was slightly sensitive to touch.

Endoscopic examination of the nose revealed in the left nasal fosa a large tumor mass of a dirty gray color, with whitish patches here and there, giving it a mottled appearance; it was of firm consistency and did not bleed readily on contact. This tumor almost entirely filled the left nasal fossa, completely concealing the middle stage, and did not retract after the application of a cocaine-epinephrine mixture. Anterior rhinoscopy did not reveal the root of the tumor, but on posterior rhinoscopic examination the tumor pedicle was seen to come out of the olfactory fissure.

Radiography on Oct. 22 showed that the frontal and maxillary sinuses were free and that the left nasal fossa was the seat of a tumor mass of considerable size. The tomograph then requested gave us the following results: Tomogram 1, carried out in a position well towards the front, 2 cm. from the anterior wall, showed a very opaque clouding of the left nasal fossa. Tomogram 2, at a distance of 3 cm., revealed a very opaque clouding, with slightly lobulated inferior contours, completely filling the left nasal fossa. Tomogram 3, at a distance of 4 cm., showed that the

lower portion of the tumor clouding was effaced, but the upper portion was still visible. Tomogram 4, at a distance of 5 cm., showed the tumor clouding to be completely effaced; the ethmoidal cells and the maxillary sinuses were clear.

Olfactory examination (Börnstein's method) revealed anosmia of the left nasal fossa and hyposmia of the right. Examination of the sense of taste gave normal results. Neurological and ophthalmic examination revealed nothing of interest. A general examination showed the heart and lungs to be normal. Blood pressure was 120/80. The electrocardiogram was quite normal. The spleen and liver were not palpable.

Laboratory examinations gave the following results: The urine was normal; the blood presented a slight lymphocytosis (red corpuscles, 4,860,000; hemoglobin, 95% white corpuscles, 5,700, with 42% polynuclear neutrophiles, 4% polynuclear eosinophiles, 2% basocytes, 43% lymphocytes, 8% monocytes, and 1% plasmocytes); blood urea was 0.32%; blood sugar was 0.72%, and the Bordet-Wasserman reaction was negative.

On Oct. 29 we removed the major portion of this tumor, which had developed in the region of the olfactory mucosa.

Histological Examination.—The excised tumor was fixed in Bouin's solution and entirely included for histological study (Examination No. 435. 70 I. P. S.) after various staining processes.

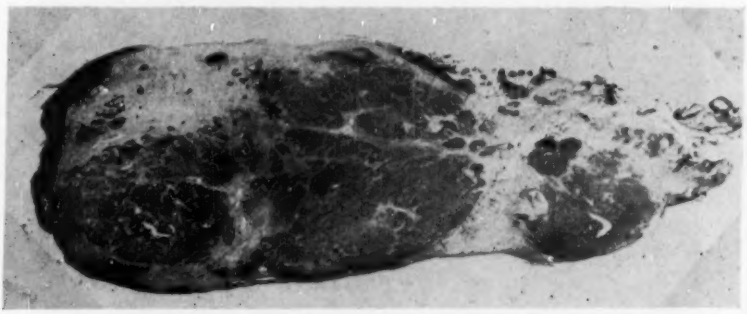

Fig. 1.—Over-all view of the tumor (median, longitudinal section) under magnifying-glass, showing the polypoidal structure of the neoplasm and its narrow pedicle (inserted in the olfactory fissure). The tumor is covered with a fibrinoleucocytal exudation. Hemalum, erythosin, and saffron stain; × 5.5.

Under a magnifying glass a median longitudinal section of the endonasal mass showed a formation of polypoid structure, with a short narrow pedicle, which was almost entirely covered by a fibrinoleucocytal crust. This magnification showed the mass to be composed of compact cellular sheets of an obviously tumorous nature, closely packed, one against another (Fig. 1).

A weak or medium magnification revealed a distinctly pseudoepithelial appearance of the tumor. It was formed of cells or large irregularly arranged cellular spars. A certain number of formations had a clear outline, perfectly marked by more or less distinct fibers of collagen (Fig. 2). More frequently, however, the outline was indistinct, the cells being separated from the ordinary conjunctivovascular stroma by a fairly thick sheath composed of elongated elements generally pale in color (Fig. 3).

The pseudoepithelial appearance of certain portions of the tumor became even more striking under powerful magnification (Fig. 4). Indeed, certain of the tumor masses had small rounded true cavities, optically empty, around which the cells were regularly arranged. These cells were tall and cylindrical, never ciliated, and the outline of the illuminated and lateral faces was distinct; the inferior portion, on the contrary, disappeared in the adjoining tumor tissues. The cavities formed never contained the slightest secretion, and the test for mucin by Mayer's mucicarmine stain was entirely negative. Therefore these were not glandular or glanduliform cavities but characteristic neuroepithelial cavities, "neuroepithelial rosettes" (Fig. 5). The interpretation was confirmed, moreover, by the disposition of the tumor tissues other than those directly

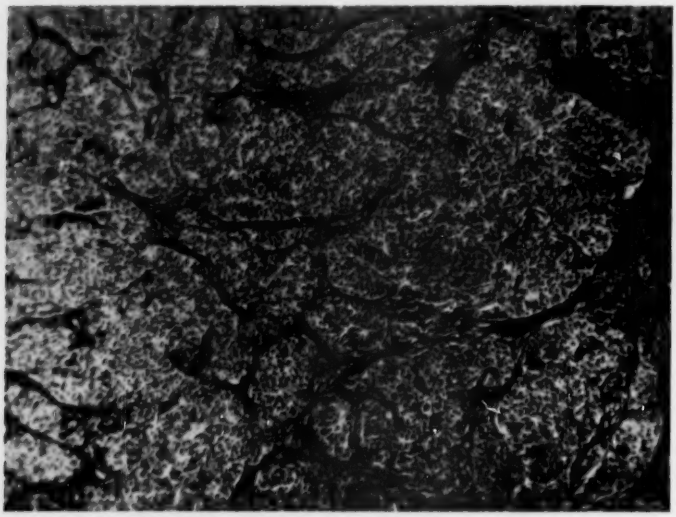

Fig. 2.—Cells and connective spars of the tumor, of pseudoepitheliomal appearance, clearly outlined by collagen fibers. Masson's trichrome stain; × 100.

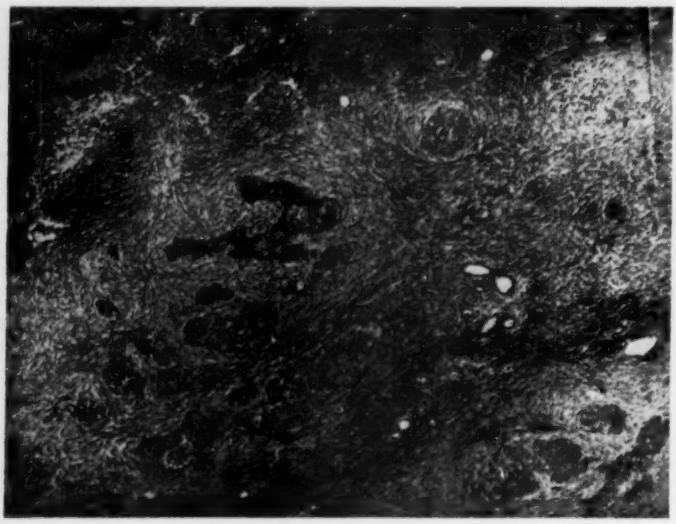

Fig. 3.—Tumor cells separated from the conjunctivovascular tissue (clearly visible as a dark trace in the top left of the figure) by a cellular "sheath," fairly broad and much lighter in color than the compact cellular groups which it encloses. Masson's trichrome stain; × 42.

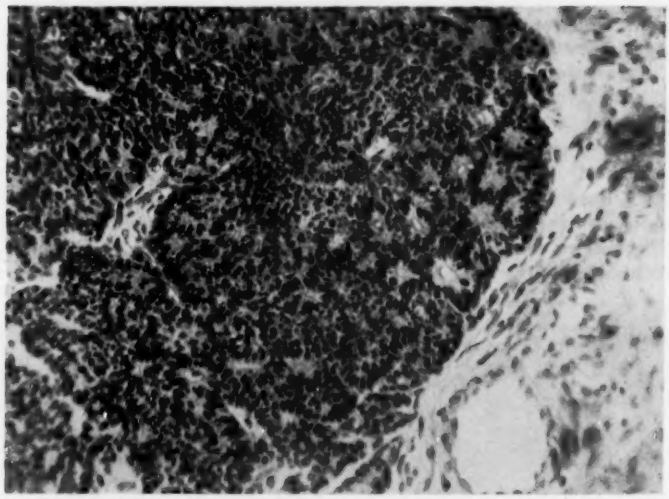

Fig. 4.—Structure of the solid portions of the tumor. In the middle of the compact zones can be perceived true cavities (right) around which the cells are regularly arranged. Hemalum, erythrosin, and saffron stain; × 180.

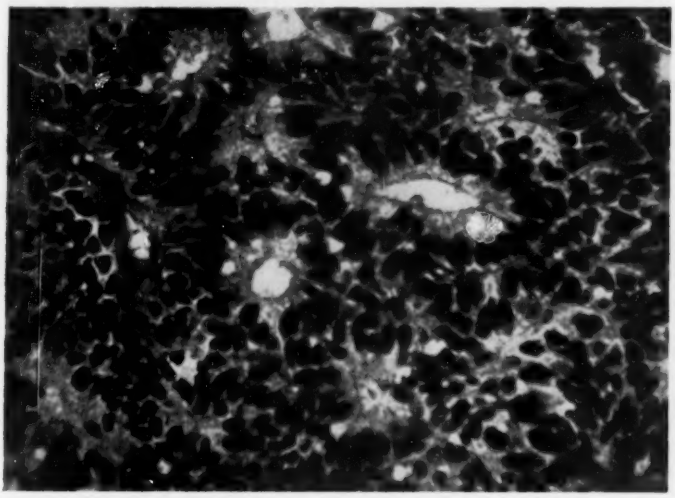

Fig. 5.—Higher magnification of portion of Figure 4. True neuroepithelial rosettes (tall cylindrical cells with distinct outlines at the apex and on the sides, the inferior portion of the cellular body disappearing in neighboring elements) and neurospongial formations (protoplasmic mingling, without cellular differentiation, scattered with nuclei) with occasional arrangements in full neurospongial rosettes (extreme left of figure). Masson's trichrome stain; × 470.

adjoining the true cavities. Indeed, the tissues in this case no longer showed any cellular division; they constituted, on the contrary, a finely reticulated sheet of proptoplasm, with no trace of collagen and with numerous scattered nuclei, either round or ovid, in a very dense chromatine pattern. In the center of this mass occasional "full rosettes" were distinguished, whose centers were composed of delicate felted protoplasm around which were arranged a crown of nuclei (Fig. 5). There could be no doubt that we were here concerned with a neurospongial evolution typical of the tumorous neuroepithelium. These neurospongial zones constituted, moreover, the essential portion of the cellular and the solid tissues of the tumor.

In the regions surrounding the neuroepitheliomal and neurospongial accumulations the tumorous neurospongium continued its nervous evolution. It thereby formed light-colored "sheaths" or "collarettes" bordering the cells, composed of less embryonic matter, more differentiated than the neurospongium. In these areas, the fairly voluminous fusiform tumor elements with large relatively light-colored nuclei, occasionally vesicular and nucleolated, were almost all individually sheathed in a very thin coating of a special folated collagen, visible only on staining

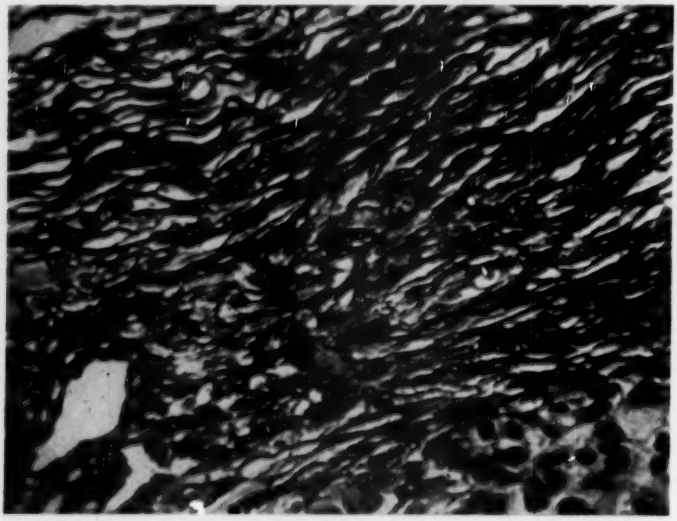

Fig. 6.—Powerful magnification of a sheath enclosing a neuroepithelial and neurospongial cell showing a far more developed nervous differentiation. The spindle-shaped elements, each bordered by a delicate frame of foliated collagen, are the result of the neuroblastic evolution of the tumor. Masson's trichrome stain; × 470.

with Masson's trichrome stain (Fig. 6). These aspects bore witness to the neuroblastic potential of the tumor. Occasionally encountered were fairly thick bundle arrangements, in which the nerve elements were stretched out in long protoplasmic ribbons or fibers. In these neuroblastic zones, abnormalities were very frequently encountered and were of great importance (giant cells, occasionally multinuclear, and abnormal mitoses, multipolar and abortive). Staining with Mallory's phosphotungstic acid hematoxylin revealed the definite absence of all glial fibers.

Therefore, to summarize, we were concerned with a malignant embryonic nerve tumor of the type encountered in the central nervous system—a neuroepithelioma subject to various differentiations, especially neurospongial and neuroblastic, the latter resulting in the formation of bundles of true nerve fibers. Consideration of the histopathological indictions combined with the clinical characteristics of the origin and endonasal development of the tumor (absence of any cerebral endocranial tumor) from the olfactory mucosa indicated a diagnosis of olfactory esthesioneuroepithelioma. The close relation between the tumor we are considering and that of Berger's original case was particularly striking.

Clinical Development.—Since we were concerned with an exceedingly malignant but in all probability exceedingly radiosensitive neoplasm, we decided to administer x-ray therapy. Between Nov. 13 and 28, 1947, the patient was given a total dosage of 6,500 r. On Nov. 28, burns of the face and nasal mucosa obliged us to discontinue radiotherapy.

In January, 1948, the nose was in excellent condition and the result of a check biopsy (Examination No. 446, 12 I. P. S.) was negative. Check roentgenograms confirmed the clinical disappearance of the tumor of the left nasal fossa but indicated that an invasion of the left frontal sinus was to be feared. We therefore performed an investigatory trepanation of the left frontal sinus; our fears were fortunately unconfirmed; the mucosa was histologically normal (Examination No. 447 I. P. S.).

The patient, reexamined at regular intervals, is in excellent condition, both locally and generally, and has remained completely cured for the last five and a half years. A curious feature is that examination of the olfaction shows that the sense of smell has been completely regained.

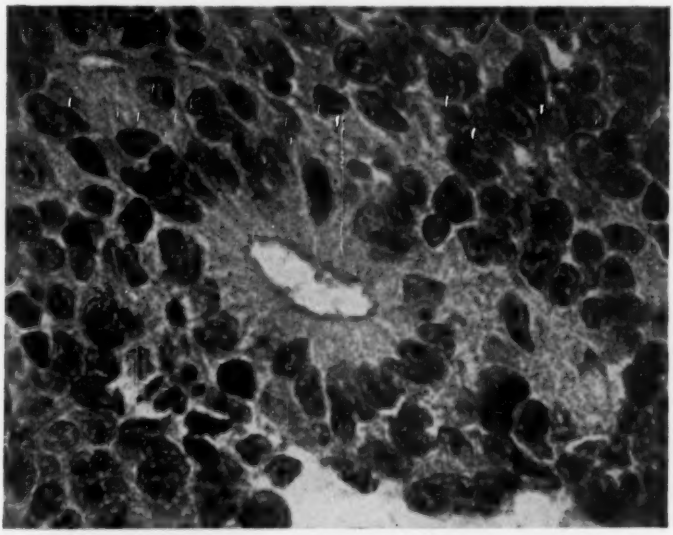

Fig. 7 (case 2).—Powerful magnification of a neuroepithelial rosette with a true cavity, limited by the apical pole of the bordering cells, which is clearly shown by a true apical strip (center). A further smaller neuroepithelial rosette is visible in the bottom right-hand corner, in the middle of the neurospongial network; × 745.

Case 2.—*Clinical Observations.*‡—Georges, M., a 63-year-old man, consulted Dr. Neiner May 31, 1932, for rhinitis and nasal obstruction of some months' standing, which had developed painlessly.

Rhinoscopic examination disclosed a considerable deviation to the left of the nasal septum and a polypoid degeneration of the mucosa of the left middle turbinate bone.

Two months later a submucosa resection of the septum was performed and the left ethmoid bone was curetted. The polypoid proliferation, of distinctly encephaloid consistency, had its real origin in the olfactory zone of the nasal septum, at which point its consistency was considerably diminished.

Histological Examination.—The tumor, removed by fragmentation, was examined histologically after being fixed in Bouin's solution (No. 115, OI I. P. S.).

The general arrangement of the tumor was cellular, the cells being compact and with a large number of small dark-colored ovoid nuclei. Powerful magnification showed conclusively that

‡ Furnished by Dr. Neiner, of Colmar.

the major part of the tumor was composed not of distinctly individual cells but of a sort of felted cytoplasm, more or less dense and dotted with nuclei. At certain points there appeared in this undifferentiated mass true nonsecretory cavities, of the true neuroepithelial rosette type, with a regular arrangement of the cells, whose apices and lateral faces were perfectly differentiated (Fig. 7).

In the portion surrounding certain cells, the elements of the tumor became more prolific and invaded the connective tissue, forming typical neurofibrils, revealed by staining with Mallory's hematoxylin stain.

Conclusion.—We were here concerned with a nerve tumor of the nasal fossae, of the classic olfactory esthesioneuroepitheliomal type, with neurospongial and, more exceptionally, glial differentiations.

Clinical Development.—Despite warnings as to the seriousness of his condition, the patient refused radiotherapy. Six months later, his doctor, answering an urgent call to his bedside,

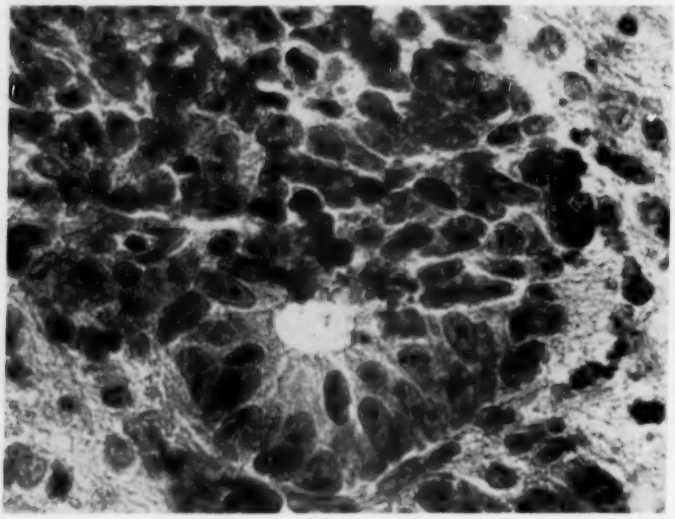

Fig. 8 (case 3).—True neuroepithelial rosette situated on the edge of a typical neurospongial cell in which a few mitotic nuclei can be discerned; × 745.

found the patient in a restless dazed condition, presenting obvious symptoms of cerebral compression, with a slow strained pulse rate.

The patient was immediately transferred to a clinic. Rhinoscopic examination revealed a recurrence of the tumor in the ethmoid region and in the upper part of the septum.

Deep and "energetic" x-ray therapy was administered for three weeks (dosages unknown). At the end of the treatment all the clinical symptoms of cerebral compression had disappeared, and after an interval of three months endonasal examination showed the complete disappearance of the tumor.

Our second observation confirmed the extreme radiosensitivity of the olfactory esthesioneuroepithelioma. Unfortunately, this patient did not remain under observation.

Case 3.—This patient consulted Dr. Bartheleme, of Colmar, for an infiltration of the mucosa of the nasal septum with complete destruction of the nasal bone, a lesion reminiscent of a syphilitic gumma.

Histological Examination.—The tumor, of distinctly cellular structure and frequently displaying a perithelial arrangement, was composed of groups of almost completely undifferentiated

cellular elements. In this cytoplasmic sheet, scattered with oval nuclei with fine often mitotic chromatin, were present fairly numerous true typical neuroepithelial cavities (Fig. 8). The neurospongial developments were rare, and other nerve developments nonexistent.

Conclusion.—This was a case of undifferentiated nerve tumor of the olfactory esthesioneuroepithelomal type, with no other nerve development.

CASE 4.—Joseph Z., a 36-year-old metallurgical worker was sent to the Strasbourg Ear, Nose, and Throat Clinic by Dr. Heckel, of Thionville, with a diagnosis of a malignant tumor of the right nasal fossa.

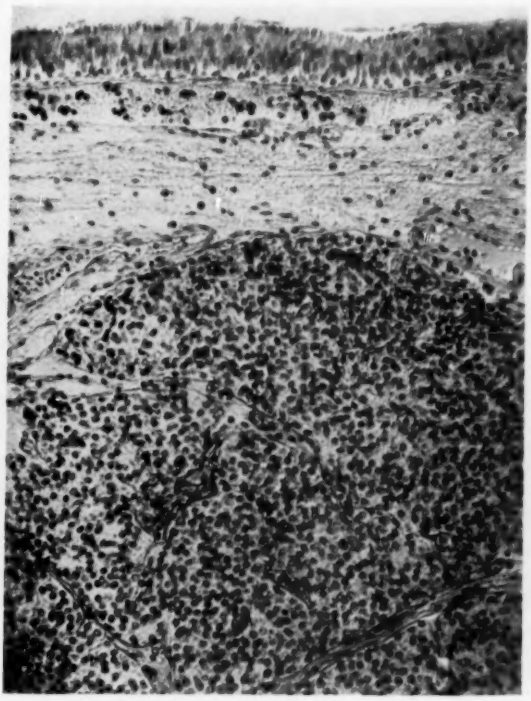

Fig. 9 (case 4).—Moderately magnified view of the tumor developing in the chorion, pushing back the ciliated cylindrical epithelium of the nasal mucosa. Note the cellular structure and the clearly defined limits of the tumor formations at their point of contact with the connecting tissue, giving the tumor a pseudoepithelial appearance; × 152.

The patient's hereditary and previous personal medical history revealed nothing of note. There was no previous history of disease of the ears, nose, or throat.

At the beginning of 1937, the patient complained of an obstruction of the right nasal fossa, with nasal discharge. There were neither nasal hemorrhage nor head pains. A few months later there was a purulent blood-tinged mucous nasal discharge, accompanied by complete anosmia and frequent but irregular pains above the right orbit.

In October, 1937, Dr. Heckel performed an endonasal operation, which was followed by a temporary improvement in the patient's condition. But in January, 1938, the obstruction of the right nasal fossa reappeared. In the months which followed, there was a renewal of the pains above the right orbit; the patient noted a considerable amount of pus coming from the right nasal fossa. Since, in addition, he experienced considerable and repeated nasal hemorrhages, he

again consulted Dr. Heckel who, on June 18, 1938, took a biopsy specimen. The pathologist who carried out the histological examination gave the following report: "Fragment of nasal mucosa completely invaded by a malignant tumor showing the structure of a cellular epithelioma with cells relatively light in color. It is probably a tumor of glandular origin. The mucous chorion surrounding the tumor is exceedingly vascular, and numerous vessels have been invaded by cancerous processes. The prognosis, based on the above evidence, is not encouraging."

On June 27, 1938, the patient entered the Strasbourg Ear, Nose, and Throat Clinic. The patient was a man 36 years of age, in sound physical condition. Examination of the ears, nose, and throat revealed no deformation of the nasal pyramid, but the right nasal fossa was completely obstructed by a hard pinkish mass, bleeding readily, partially ulcerated, and seeming to have its origin in the ethmoid. All general examinations were negative. The tumor mass was also visible by posterior rhinoscopy via the right posterior orifice of the nasal fossa.

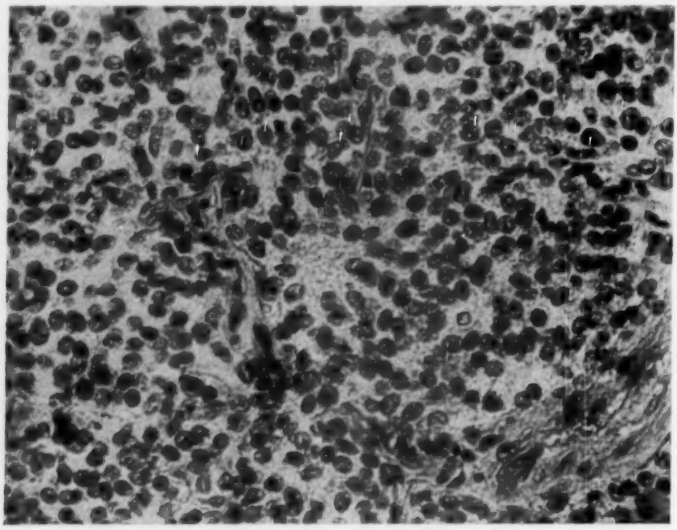

Fig. 10 (case 4).—Fairly powerful magnification of the tumor, showing its typical neurospongial nature with a reticulated fibrillary cytoplasmic mass scattered with nuclei, in which full neurospongial-type rosettes, without central cavities can be seen (center); × 320.

July 1, 1938, Professor Canuyt performed the surgical transmaxillonasal ablation of the tumor, with a local anesthetic. The tumor had invaded the whole of the right nasal fossa, maxillary sinus, and ethmoid.

Histological Examination.—Examination of the ablated tissue showed a tumor which had raised the ciliated cylindrical respiratory epithelium of the pituitary without ulcerating it; its general structure was of a trabeculocellular nature, with extremely distinct outlines near the connective tissue, i. e., it was definitely pseudoepitheliomal (Fig. 9).

Powerful magnification showed both cells and connective processes to be formed of a cytoplasmic mass in which were perceived elongated elements of a neuroblastic type; quite frequently the nuclei, instead of being irregularly arranged, were grouped around spherical anuclear spaces filled with a fine felted cytoplasm, thus constituting neurospongial rosettes (Fig. 10). A few true neuroepithelial rosettes were found, but these were not numerous and remained generally small.

Conclusion.—This was a nerve tumor of the nasal fossae of the olfactory esthesioneuroepitheliomal type, with frequent neurospongial differentiations.

Clinical Development.—From July 15, 1938, to August 10, x-ray therapy was administered to the patient (total dosage, 4,500 r). On Aug. 20 the patient left the Ear, Nose, and Throat Clinic in excellent condition. Unfortunately, we have since been unable to keep this patient under observation.

COMMENTARY

The four cases observed call for a certain number of comments.

From the theoretical point of view, it is both interesting and important to note that true nerve tumors can originate from a true entodermic formation.

From the clinical point of view, the symptomology of the nerve tumors of the olfactory organ is quite ordinary and in no way specific. The nasal obstruction, nasal hemorrhages, head pains, and anosmia are quite usual.

Clinical diagnosis is impossible, and any slightly unusual or suspicious polypoid formation in the nasal fossae should be subjected to histological examination.

Histopathology.—The diagnosis can present certain difficulties of interpretation, particularly to the histologist unaware of the existence of these nerve tumors. Our Case 4 is an example demonstrating these difficulties of microscopic diagnosis, since the examination of the first biopsy led the histologist to postulate a diagnosis of glandular epithelioma, being missed by the distinct outlines of the cells in the region of the stroma. The case of Bosc and Browaeys [14] gave rise to four successive histological interpretations, all different. Indeed, in addition to the pseudoepitheliomal forms, there exist pseudosarcomal forms with spindle-shaped elements, or with lymphocytoid elements, or even polymorphous forms. A rigorous histological technique with, especially, a study of the collagen, using Masson's trichrome stain, can be of great help.

Finally, from the therapeutic point of view, the extreme radiosensitivity of the true olfactory esthesioneuroepitheliomas should be stressed, contrary to certain opinions, such as those of Terracol.[18] In our cases (Cases 1, 2, and 4) we observed a spectacular disappearance of the tumor under the influence of moderate doses of x-rays. In Case 3 we have no therapeutic indications whatsoever.

In addition to this extreme radiosensitivity, the malignancy of these tumors is certainly attenuated. Their growth is relatively slow, and, even though they are capable of destroying neighboring bone structures, the majority of writers have noted the absence of metastatic propagation. In one case only, that of Seaman,[10] did the patient, eight years after the beginning of x-ray therapy, develop metastases of the cervical ganglion (equally radiosensitive) and then mediastinal and pleural metastases.

The survival following radiotherapy is often considerable, being more than five years in Case 1 (and the patient is still in good health). We have been unable to follow up the other cases. Survival after surgery and radiotherapy is equally considerable, being more than six years in the case of Huet,[11] more than four years in the cases of Martin, Dargent, and Gignoux,[9] and four years in the case of Stout § after radiotherapy only, there having been a recurrence of the tumor after surgical ablation.

We personally believe the preferred method of therapy to be surgical ablation of the tumor mass obstructing the nasal fossae followed by a course of high-dosage irradiation.

§ Cited by Seaman.[10]

SUMMARY AND CONCLUSION

Four personal anatomoclinical observations of true olfactory esthesioneuroepitheliomas of the nasal fossae, of the type reported by Louis Berger, are reported. Neurogenic tumors of the nasal fossae originating in the olfactory organ are admittedly infrequent, but it is probable that their rarity is partially due to failure to recognize them (owing to the possibility of histological confusion with glandular epitheliomas or sarcomas). We would stress the attenuated malignancy of these tumors, their extreme radiosensitivity, and the considerable length of survival of the carefully treated patient.

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GLOMUS JUGULARE TUMOR

Report from Puerto Rico

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AND
J. E. TAVERAS, M.D.
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LITTLE is known about tumors of the glomus jugulare (nonchromaffin paraganglioma), a condition which most otologists know only by reference, if at all, but which is not as rare as it is thought to be by Dr. Harry Rosenwasser,* who reported the first case diagnosed as such in 1945 "as a pathologic curiosity." Lately there have been more reports in the periodicals about glomus jugulare tumors, and we believe that it is rather important that one be on the look-out for these cases, some of which might not be as benign as is the general belief of the very few who have seen these cases.

The first man we met who could tell us of personal experience with tumors of the glomus jugulare type was Dr. Jose Gross,† Professor of Otology of the University of Habana School of Medicine, who had had experience with a very malignant case in which there was destruction of the petrous bone and secondary abscess of the left lobe of the cerebellum. The pathologic diagnosis of this case, after the last of several surgical interventions, was angiomatous granulation tissue. The final diagnosis of the tumor as a tumor of the glomus jugulare was done by Dr. Raffaele Lattes from some autopsy material. During the course of the disease, this case was complicated by palsy of the left 9th, 10th, 11th, and 12th cranial nerves and later by facial paralysis, and, as mentioned before, by destruction of the petrous bone, all due to pressure of the tumor, and a cerebellar abscess due to secondary infection. This case was treated by radiotherapy as well as surgery, with hardly any benefit. This case was reported by Lattes and Waltner [1] in their series from the Surgical Pathology Laboratory, Columbia University, New York City.

Our case is therefore the first case of glomus jugulare tumor to be reported as such in the West Indies, and probably in Latin America, with a clinical and pathological study of the case as such.

REPORT OF CASE

The patient, I. M. R. (No. 34128), was a 60-year-old white, poorly nourished woman from the rural area of the western part of the Commonwealth of Puerto Rico. She was sent to the Aguadilla District Charity Hospital March 27, 1951, with the complaint of right-ear disease

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* Rosenwasser, H.: Personal communication to the authors.

† Gross, J.: Personal communication to the authors.

for the past six months and was referred to me the following day. The history of the case is irrevelant except for the frequency of head colds and her statement that 10 years previously she felt, one day, as if something had entered her right ear and occluded it and that since then, she had became progressively hard of hearing. She noticed nothing else except that about one year or so before admission the right ear began to suppurate and give off a watery discharge and that then hearing became greatly reduced in that ear. About six months before her admission as a patient, the right ear started to feel as if it were plugged with something.

Local examination revealed pus in the right ear canal, which, when cleaned out, opened to view a polypoid granuloma, which, on being lightly scraped, began to bleed to the extent that it had to be lightly packed to be controlled.

Remembering the advice of Dr. George Shambaugh Jr.—"not to pull on an ear polyp, because you do not know to what it may be attached"—I (A. P. T.) requested an x-ray study and decided to do an exploratory mastoidectomy.

X-ray report of April 6, 1951, was as follows: "Roentgenogram of the mastoids reveals a homogenous opacity of the right mastoid. Impression; mastoiditis, right."‡

Routine laboratory studies, including Kahn and Kline tests, were negative.

On April 18, 1951, with ether anesthesia, an endaural exploration of the mastoid and middle ear on the right side was attempted with the following findings: a large warty growth com-

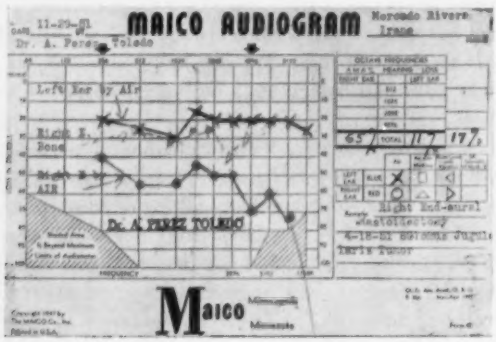

Fig. 1.—Audiogram taken seven months postoperatively.

pletely filled the right ear canal. The mastoid cavity was entered through the Shambaugh endaural approach, and the mastoid cavity and antrum were found to be completely filled with granulation-like tissue, some inflammatory debris, and papillomata. All the tissue was very friable, and some bloody oozing made the operation more difficult. Feeling that I might be dealing with a carcinoma, because of a case I had had some time before, which turned out to be a carcinoma of the ear, I decided to clean the mastoid as far as possible without risking profuse hemorrhage. The mastoid was cleaned down to the aditus without entering the tympanic cavity proper. I had the impression at the aditus that I had pulled some of the pathologic tissue from the middle ear. This is very possible, for later the drum membrane healed completely and is still intact today.

The general postoperative condition was not satisfactory as there was cough and nausea for some days; the dressing was blood-tinged, but there was no evidence of hemorrhage. Otherwise the postoperative course was uneventful. The patient stayed in the hospital a little over two weeks because of the cough and for general care, but no postoperative hemorrhage occurred. By the end of one month the ear was healing satisfactorily and was almost dry.

For the next few months the ear healed very satisfactorily, with the growth of an intact drum membrane, but still the patient complained of deafness and buzzing in the right ear.

X-ray report of July 20 (about three months postoperatively) was as follows: "Postoperative roentgenogram of the mastoids revealed an area of rarefaction in right mastoid."

‡ Dr. J. N. Cardona Quiñones, radiologist.

An audiogram was taken Nov. 29, 1951 (seven months postoperatively) is seen in Figure 1. The audiometric curve of the left ear is within normal limits for practical hearing; but the audiometric curve of the right ear shows a loss of 45 to 55 db. for the most important frequencies, going down to a 60 and 70 db. loss from the 4,000 to the 8,000 frequencies. The bone conduction of the right side ranges from 25 to 40 db., which is a rather good bone conduction for the case. For clarity, the bone conduction for the left side is not shown in the audiogram, but the reaction to Weber's test was so marked to the right that bone conduction of the left mastoid was heard almost equally loud on both sides.

I have been observing this patient every two or three months ever since and expect to have her come for a radical mastoidectomy at the first sign of trouble, but so far she is doing quite well.

The drum-membrane condition is worthy of description. It is an intact drum membrane, characterized by bulgings separated by well-defined sulci. There is one large posterior bulging and two smaller anterior bulgings, one above and one below. All of these bulgings pulsate

Fig. 2.—Portion of tumor lobule composed of syncytium-like masses of epithelioid cells. The islets or bands of tumor cells are surrounded by richly vascularized trabeculae (× 220).

synchronously with the pulse; the anterior ones pulsate in and out with the pulse; the large posterior one pulsates in a semipendular movement forward and backward with the pulse, giving the impression that it carries the malleus with it forward with systole and backward in diastole. The movement is really behind the drum membrane. The whole drum membrane is homogenously dull red, but no vessels can be seen in it as in an acute inflammation of the ear. The last time I saw the patient, in December, 1952, the drum membrane was in about the same condition that it has been for the past year and a half except for a slight increase in the size of the anteroinferior bulging, which seems to be trying to creep underneath the lining skin of the ear canal floor.

Tuning fork test showed air conduction greater than bone conduction in the left ear and bone conduction greater than air conduction in the right ear, with hardly any air conduction at maximum intensity. The reaction to Weber's test was strongly to the right, denoting good cochlear function on the right side; deafness was prominently of the conductive type.

I am waiting for complications in the patient's family to clear up before making a radical removal of the tumor from the tympanic cavity, which I hope to do before pressure of the tumor causes any destruction in the temporal bone.

Pathology.—Pathological report by Dr. J. E. Taveras was as follows: Gross examination showed seven separated portions of tissue ranging in dimension from 0.5 cm. to 1.3 × 10 × 0.5 cm. The external surface was uneven and brownish in color. On section the tissues were moderately dense and rubbery in consistency. The cut surface was composed of irregular areas

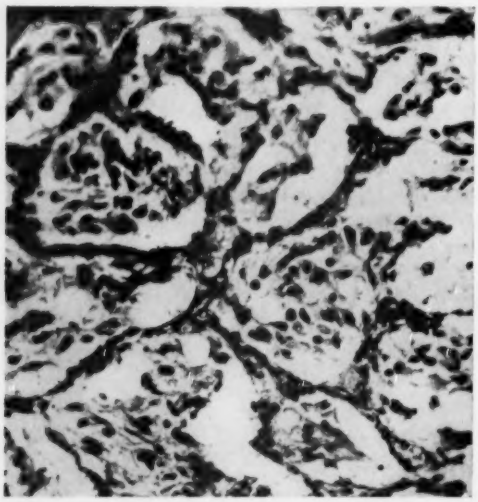

Fig. 3.—Same area of tumor that is seen in Figure 2 but at a greater magnification (× 450).

Fig. 4.—Low magnification of superficial area of the tumor including the covering of squamous-cell epithelium. A rather dense leucocytic infiltration is present in the zone where the tumor islets are shrunk and are seen somewhat replaced by fibrous tissue (× 100).

of light yellowish, grayish, or chocolate color. The specimen was received in formalin fixative. All the fragments were totaly embedded.

Microsections included portions of a tumor composed of ill-deliniated lobules formed by islets, clumps, and short bands made up of polygonal or epithelioid cells showing acidophilic, finely granular cytoplasm. The nuclei were relatively small, darkly stained, or vesicular. The islets

and other tumor formations were separated by more or less thick trabeculae, richly vascularized and showing capillary channels lined by a thin wavy endothelial wall. Many of these channels were dilated with blood. In some areas the tumor cells were arranged in syncytium-like formations. Some of the tumor lobules exhibited degenerative changes, and groups of cells appeared shrunken, their cytoplasm intensely stained with eosin. The nuclei were pyknotic or karyorrhectic. In other instances the degenerative cells contained in the cytoplasm showed multiple punctiform vacuoles, frequently associated, forming small irregular intracytoplasmic cavities. In these same areas the cells were frequently fusiform or polygonal, as previously described. In the areas of more advanced degeneration, the tumor cells were seen even more elongated and strongly acidophilic, and in the stroma the capillary network of the tumor was more prominent. No mitotic figures were seen. The surrounding stroma of the tumor was composed of dense connective tissue showing areas of edema beneath the epithelium which covered the outside of the specimen. This epithelium was detached in many places. Foci of lymphocyte and plasma cell infiltration were seen here and there in the sections.

In many places of the tumor there was a partial replacement of the tumor by connective tissue. Areas of hemorrhage were prominent in the outer portions of the tumor fragments.

The specimen was partially covered by a stratified squamous epithelium that was slightly thickened in places and atrophic in others. The epithelium was covered by a thin keratinized membrane, beneath which a granular layer composed of three or four rows of cells had developed. This tumor can be compared morphologically with that found in the carotid body and was similar in appearance to that described by Rosenwasser in 1945.

COMMENT

The normal glomus jugulare structure was first described by Dr. Stacy Guild § as a "hitherto unrecognized tiny structure in the region of the ear." It is similar to the carotid body, located in the adventitia of the dome of the jugular bulb below the floor of the middle ear near the ramus tympanicus of the glossopharingeal nerve. Guild called it the glomus jugularis and the name was changed to glomus jugulare by Lattes and Waltner,[1] and has been known as such since then by most people. It was not until 1945 that Dr. Harry Rosenwasser reported his case and pointed to the possibility of its relationship to the glomus jugulare described by Guild in 1941, this being the first case of glomus jugulare tumor to be reported in the literature as such.

No mention of glomus jugulare tumor is made in books (textbooks or otherwise), but excellent articles describing this pathologic condition have been presented by Winship and Louzan,[2] Lattes and Waltner,[1] and several others in the various medical periodicals, especially *Cancer* and the A. M. A. Archives of Otolaryngology.‖ Cleary [3] reported what he thought was the 61st case; but according to a personal letter from Winship (1952) there have been 65 cases collected from the European and American reports. Thus this case of ours is probably the 66th or 67th case reported in the world and one of the very few that are still under observation for further study.

On passing through New York recently, I (A. P. T.) showed two slides (prepared by Dr. Taveras) of this case to Dr. Rosenwasser, Dr. Lattes, and others for further confirmation. Lattes authorized me to quote him in his report. "I agree that the tumor is a nonchromaffin paraganglioma, probably arising from the glomus jugulare."

§ Guild, S. R.: Personal communication to the authors.

‖ References 2 to 5.

SUMMARY AND CONCLUSIONS

This is the first clinical and pathological report of a case of glomus jugulare tumor from the West Indies, and probably from Latin America. One case from Cuba is now known to have been a case of glomus jugulare tumor of the middle ear, but this case was never diagnosed, either clinically or pathologically, as such until some time after death from some postmortem material sent to Columbia University Pathological Laboratory. This case was finally included in Lattes and Waltner's [1] series reported in 1949 as their eighth case. It is the belief of Lattes that "among the peritheliomas, adenomas, and hemangioendotheliomas reported in the literature, there are tumors of this kind."

From our case we conclude that polyps of the ear, no matter how benign they appear, should be studied conscientiously, and, by all means, microscopic studies should be done of the material obtained. Our case is one of an apparently, so far, benign neoplasm of the middle ear, characterized by polipoid or papillary formations that protruded out of the external auditory canal with secondary infection and suppurations, which healed after an incomplete enucleation. This resulted in an intact drum membrane some time after operation, which still pulsates synchronously with the pulse. The main symptomatology was the appearance of the ear polyp formation with a tendency to bleed markedly when scraped, conductive deafness with good bone conduction, a sensation of buzzing in the ear, false healing of the drum membrane over the remaining portion of the tumor after an incomplete enucleation, and no apparent effect in the general health of the patient.

Most of these tumors have a benign pattern like this case of ours, but there have been cases reported as malignant—one with metastases to the liver, another with metastases to the neck and brain. One such tumor caused the destruction of the temporal bone, cerebellar abscess, and rapid death; others have caused paresis or paralysis of different cranial nerves by direct pressure.

This is an unpredictable tumor with some possibilities of metastases, hemorrhage, or destruction of nearby structures in the temporal bone by direct pressure. These tumors are resistant to x-ray and radium therapy, so surgery is the treatment of choice and should be done as early as possible.

Dr. R. Lattes reviewed the slides, and Dr. T. Winship encouraged us to report the case.

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TYPES OF OTOSCLEROSIS

Their Clinical Manifestations

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DIVERSIFIED types of otosclerosis are encountered clinically. The commoner characteristics of this disease include progressive deafness, tendency to bilateral involvement, family history of deafness, tinnitus, paracusis, comparatively good bone conduction, and a soft voice, which is generally well modulated.

The fulminating type of otosclerosis has an acute onset, with headache, nausea, and dizziness. Tinnitus is most disturbing, and within a few months the patient becomes almost completely deaf in both ears. Schwartze's sign—a pinkish tinge over the promontory—is apt to be present. The course of the disease is moderate in most cases. In 5 to 10 years, social inadequacy ensues when the impairment in the better ear becomes greater than 35 db. for the speech tones. To some patients with otosclerosis of moderate severity, tinnitus is the most disturbing feature. In a few instances, a psychogenic factor may be detected. Two of my patients, who complained of unbearable "clicking," "thumping," and "roaring" noises when chewing or swallowing, obtained complete relief from the fenestration operation. It appears that the mechanism of the tinnitus was the perverted relationship between bone conduction and air conduction. The restoration of good air conduction by the fenestration operation produced a masking effect on the sensitive bone conduction of these patients. In its mildest form, otosclerosis progresses slowly, with periods of limited activation and lengthy intermission. The resulting impairment, over a stretch of a decade or more, is the inability to hear clearly in a lecture room or at a meeting. Tinnitus was present in 27 of 30 such patients studied by me. All had soft, well-modulated voices, which seemed to be attributable to the patients' living in a silent world. The fact that Lempert [1] was able to ameliorate tinnitus in 90% of his fenestration cases might indicate that when the silent world becomes noisy, tinnitus is masked out. Subaudible tinnitus is physiologic. Over the stream of the labyrinthine fluids there is a "ripple," which is propagated by bodily activity. This subaudible tinnitus was rendered audible experimentally by Heller and Bergman.[2] Eighty normally hearing adults were placed in a soundproof chamber with an ambient noise level no greater than 18 db. Ninety-four per cent of these subjects developed tinnitus, which had all the characteristics of pathologic head noises, while none had ever experienced tinnitus in ordinary living conditions. It appears that the ambient noise of 35 db. or more which floods the environment has an adequate masking effect to keep the physiologic tinnitus subaudible.

Vertiginous attacks are not common in otosclerosis. About 25% of the patients recall spells of momentary dizziness when stooping. A diagnostic problem arises

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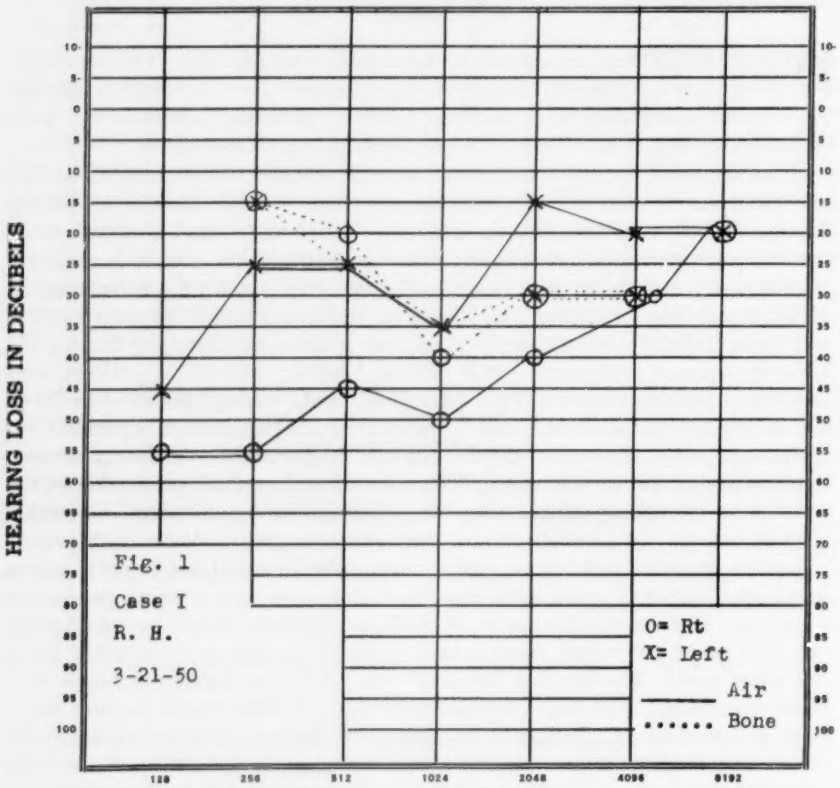
when a patient with the otologic features of otosclerosis reports seizures of vertigo. The neurologic examination may help to rule out a brain stem lesion. McKenzie [3] reports four cases of vertigo due to otosclerosis. In the differential diagnosis, he stresses the fact that normal responses to caloric stimuli plus a characteristic audiometric pattern indicate otosclerosis, whereas diminished reactivity to the caloric test suggests Ménière's disease. Hulk and Jongkees [4] performed both the caloric and the rotation test on 50 patients with clinical otosclerosis. No pathologic changes were noted after caloric stimulation employing both hot and cold water. The post-

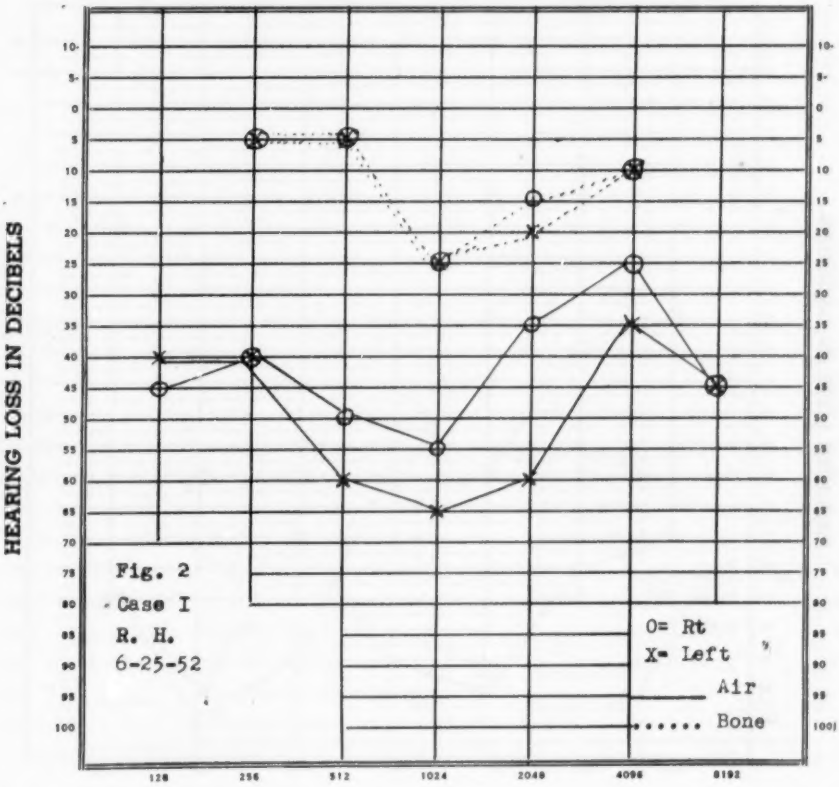


rotational reactions were marked by distinct deviations from the normal. While postrotational vertigo was diminished, nystagmus was notably prolonged. The 50 cases of these authors did not include any with vertiginous attacks. In two cases to be presented here, vertigo was present. The postrotational reactions were featured by a markedly distorted relationship between nystagmus and vertigo.

REPORT OF CASES

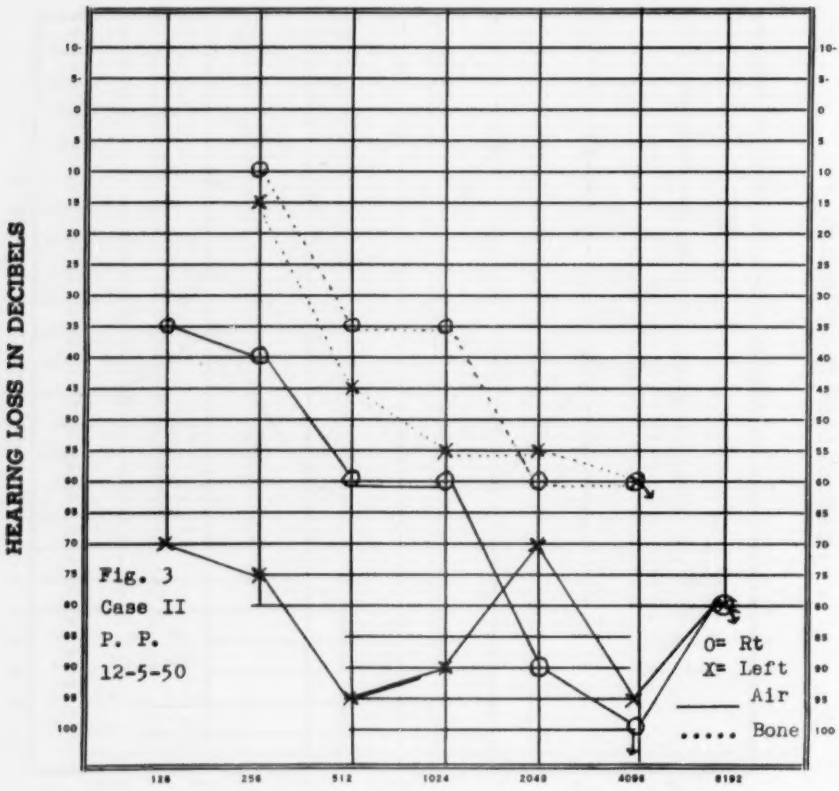
Case 1.—R. H., a white woman, aged 32, was admitted to the Jewish Hospital on March 15, 1950. Her complaints were dizziness, nausea, "thumping and droning" in the right ear, palpitation, and a sensation of "spinning," with tendency to fall to the right. The patient recalled that in August, 1946, after the birth of her child, the right ear appeared "deaf" when she lay

on her left side. For a year or more prior to her admission, she was subject to mild "dizzy spells" with a sensation of falling. Otherwise, the past medical history was irrelevant. There was a history of deafness in the patient's mother and brother. Physical examination, including otoscopy and nasopharyngoscopy, disclosed no abnormality. A neurologic consultant found no evidence of intracranial pressure or a space-taking lesion. The ophthalmologist found nothing abnormal in the eyes. Laboratory tests yielded normal results. These included x-ray of the skull, electrocardiogram, urinalysis, blood chemistry, and a complete blood cell count. The blood Wassermann reaction was negative. A spinal puncture showed normal pressure, a cell count of zero, normal chemistry, and a negative Wassermann reaction. Neuro-otologic tests gave abnormal results. The Romberg test revealed a tendency to fall to the right. Spon-



taneous horizontal nystagmus was noted on right lateral gaze only; there was none in any other direction. No spontaneous past-pointing with either arm was demonstrable. The caloric test gave normal reaction after douching for 40 seconds. The rotation test showed disproportionate reactions. When the patient was turned to the right, the duration of horizontal nystagmus was 44 seconds, while vertigo lasted 22 seconds, and immediate past-pointing to the right with either arm was 3 in. (8 cm.). When the patient was turned to the left, the duration of the horizontal nystagmus was 39 seconds as compared with vertigo of 26 seconds and past-pointing of 3 in. to the left with both arms. The audiogram (Fig. 1) obtained on March 21, 1950, showed a greater loss for the low frequencies and markedly better bone conduction for the low tones in the right ear. After completion of the preliminary studies, on March 22, 1950, the patient was discharged from the hospital, and she was referred to the outpatient

department for further study. In October, 1950, the patient went to the Lempert Institute of New York, where the fenestration operation on the right ear was performed. Partial relief from tinnitus ensued after the operation, but the dizziness, which has been continuous, is estimated by the patient to be worse than before the operation. In 1951, the patient noticed a progressive deterioration of hearing in the left ear, in which intermittent tinnitus also began to appear. There was a persistence of the sensation of "rocking" in spite of all therapy. She was referred to me on June 25, 1952. Handicapping deafness was present. The Rinne test was negative bilaterally. Figure 2 is the audiogram obtained on that date. A hearing aid fitted to the left ear gave the patient adequate hearing and considerable relief from her tinnitus.



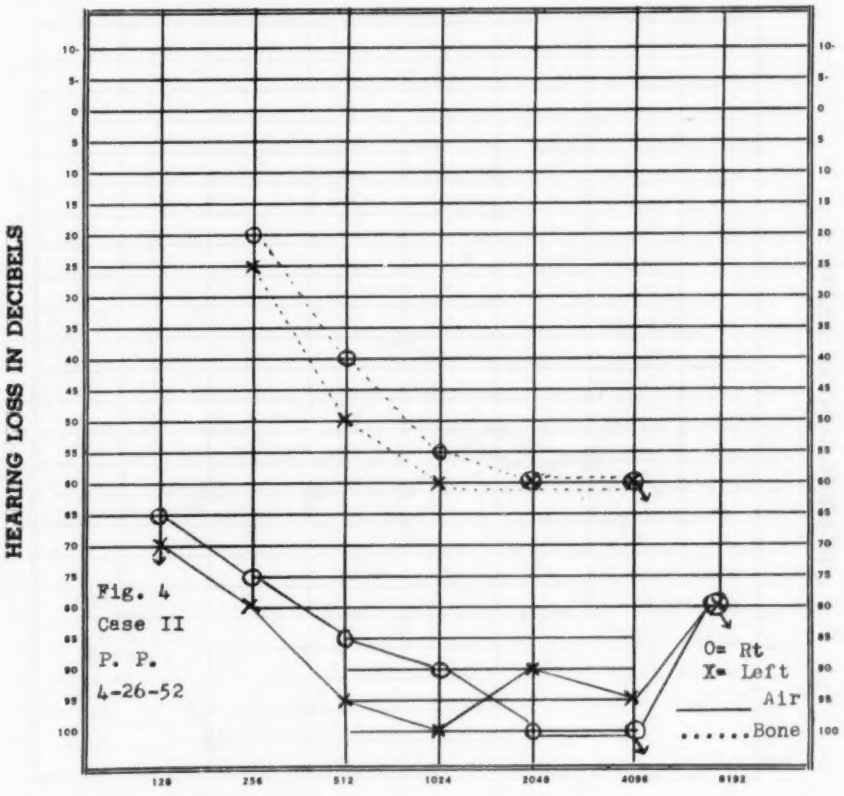
Summary.—This case appears to present irritative labyrinthitis as a feature of one form of otosclerosis. A noteworthy finding is the prolonged postrotational nystagmus. The fenestration operation gave slight relief from tinnitus but none from vertigo.

Case 2.—P. P., a white man, aged 30, was referred to me on Dec. 5, 1950. His complaint was deafness of two years' duration. It began with episodes of vertiginous attacks, unbearable noise in his left ear, and recurrent frontal headache. These episodes continued from December, 1948, to March, 1949, when the attacks of vertigo and the unbearable tinnitus ceased and the patient found that he was totally deaf in his left ear. His right ear gave him adequate hearing, although some impairment was noted. Intermittent mild tinnitus referable to both

ears was present all through the year 1949, but it increased in severity in 1950. At the time of this consultation, roaring tinnitus was present in both ears, and speech was heard only at close range.

The patient had been in good health up to December, 1948, although his wife recalls that she had noted with amazement that her husband could hear better than anyone else while traveling in the subway.

The family history disclosed that fulminating deafness was present in two previous generations. The patient's mother became profoundly deaf in her 38th year. She was able to hear only some very loud words shouted directly into her ear. His maternal grandmother and his

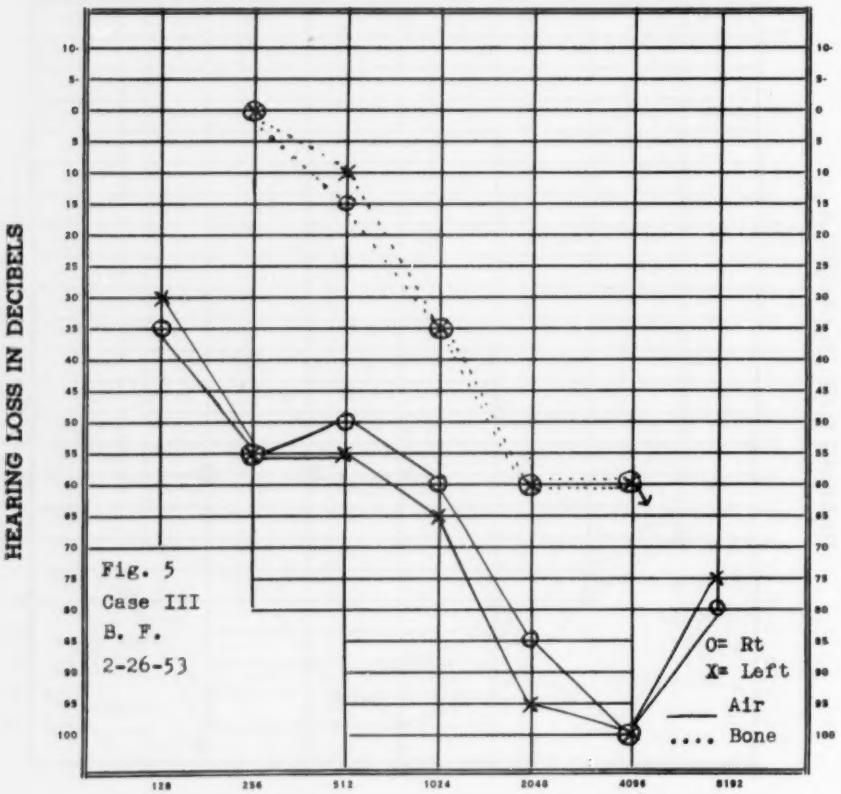


mother's only brother were almost totally deaf by the time they reached the age of 40. There was nothing in the family history to suggest a syphilitic infection. The patient's son, aged 9 years, enjoys good health.

A neurologic examination disclosed nothing abnormal. Blood serologic studies done on Dec. 7, 1950, gave negative Kahn and Kolmer reactions. Rhinoscopic and direct nasopharyngoscopic examinations revealed no significant abnormality. Otoscopy showed a pinkish tinge over the promontory in the right ear. The Rinne test, done with suitable masking, was negative bilaterally to all the tuning forks that the patient heard by both air and bone conduction. The Weber test lateralized to the right. The caloric test yielded normal responses from both ears. Figure 3 is the patient's audiogram obtained on Dec. 5, 1950.

An air-conduction hearing aid with moderate amplification gave satisfactory hearing to the right ear up to April 5, 1952, when the patient awoke with a "head full of whistles and

noises." Both ears were severely deaf. "Dizziness" was present constantly. This acute episode subsided gradually, and on April 11, 1952, the patient was able to hear through his hearing aid when the volume was increased greatly. Tinnitus of moderate severity persisted, and mild attacks of dizziness recurred several times during each day, particularly on moving the head in a horizontal or vertical plane. Occasionally, dizziness followed also the hearing of explosive sounds, and it was frequently severe after wearing the hearing aid for a long time. His walking on a hard floor without wearing the hearing aid gave him the sensation of a hammer knocking on an anvil. On April 26, 1952, otoscopy showed a pink glow over the promontory of the right ear. Positional nystagmus Grade II could be obtained intermittently. There was

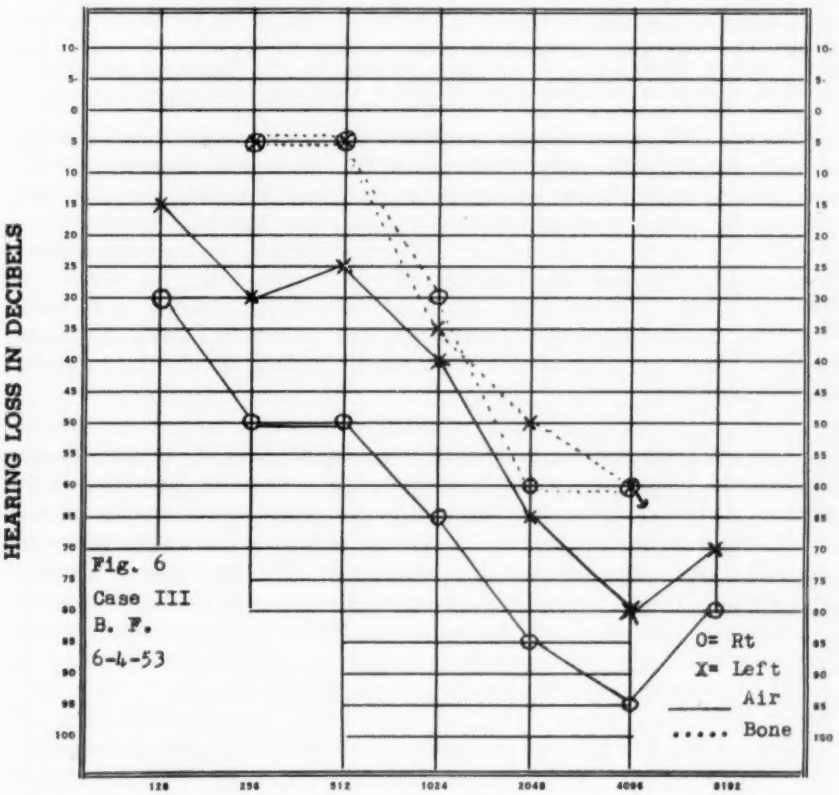


rotary nystagmus to the right when the head was swung suddenly to the left. However, it could not be reproduced each time the head was tilted into this position. Figure 4 is the audiogram obtained on April 26, 1952.

On June 6, 1953, the patient reported that dizziness and bilateral tinnitus continued and that he was still "getting by" with his powerful hearing aid. A complete Bárány test was done on this date. The caloric test yielded normal responses from both ears. The postrotational reactions showed a markedly distorted relationship between nystagmus and vertigo. When the patient was turned to the right, nystagmus to the left had a duration of 26 seconds and vertigo, 6 seconds. While the patient was turned to the left, nystagmus to the right lasted 23 seconds and vertigo, 4 seconds. The results of hearing tests done on June 27, 1953, did not show significant changes from those recorded on April 26, 1952.

Summary.—A type of fulminating deafness traceable to three successive generations is recorded. The patient's history and the audiologic pattern justify the diagnosis of clinical otosclerosis. Irritative labyrinthitis constitutes a complicating feature in this case. The postrotational disharmonious reactivity regarding nystagmus and vertigo is noteworthy. Positional nystagmus could be demonstrated inconstantly.

CASE 3.—B. F., a white woman, aged 26, was referred to the Hearing Clinic of Temple University Hospital on Feb. 26, 1953. Her chief complaint was deafness. She was also suf-



fering of bilateral tinnitus, which resembled the "rattling of a window." It was ascertained that her hearing difficulty was progressive. An impairment was noted at 10 years of age. By the time she reached 16, a hearing aid became a necessity. Tinnitus has been present since age 19. Her menstrual life began at age 11. She has an only daughter, 2 years old, and she is uncertain whether childbirth aggravated her ear condition. Her past medical history and family history are noncontributory.

Otoscopy disclosed no abnormality. Nasopharyngoscopy showed some lymphoid tissue at the midline of the posterior pharyngeal wall, but the Eustachean orifices were not obstructed. The patient spoke in a soft, well-modulated voice. It was found that when the patient was shielded from lipreading and was without the use of her aid, speech hearing was poor at a

distance of 2 ft. (61 cm.). Her hearing aid gave her excellent hearing. The Rinne test was negative bilaterally to the fork at 256, 512, and 1,024 cps. Figure 5 is the preoperative audiogram which was obtained on Feb. 26, 1953.

It was felt that this case represented a type of otosclerosis in which marked basilar atrophy was an outstanding feature. The patient was well adjusted to the use of a hearing aid on the right ear, while the audiogram showed a slightly greater loss—for speech tones—on the left side. To be sure, she was considered an unfavorable prospect (Class C) from the standpoint of selectivity for the fenestration operation. However, as her left ear was practically useless to her, it was suggested to the patient and her husband that the fenestration operation on the left ear might prove of some benefit. Upon my urging, Dr. Bernard J. Ronis performed the fenestration operation on the left side on April 23, 1953, at Temple University Hospital. I was gratified to learn that a month after the operation the patient was able to carry on a telephone conversation with the receiver to her left ear. She is also free of tinnitus since the operation. Figure 6 is the audiogram obtained on June 4, 1953. On that date, she still had to wear the hearing aid on the right ear in her daily life. The objective of restoration of serviceable hearing was not attained but considerable benefit has been brought about.

Summary.—This case presents "a half audiogram otosclerosis." There is almost complete ablation of the auditory function for the high tones. According to Brunner,[5] atrophy of the basilar portion of the cochlea is frequently associated with otosclerosis. Some recoupment of function in the viable high frequency fibers was brought about by the new physiologic mechanism created by the fenestration operation, which, at the same time, benefited all tones by by-passing the obstruction. From Shambaugh's statistics,[6] it may be inferred that the fenestration operation is effective in retarding nerve degeneration in some cases. The tinnitus in this case must have been due to otosclerosis rather than to cochlear atrophy, as it subsided after the operation.

COMMENT

A complicating condition modifies the symptomatology of any disease. In otosclerosis, the clinical picture will be affected by an abnormality prevailing in the external, middle, or inner ear. Distorted pure-tone and speech audiograms will result from a psychogenic overlay. A cochlear defect may become superimposed. Acoustic trauma, virus infection, or a cochlea-toxic drug may bring about neural degeneration. This presentation is concerned, however, with the basic differences of the natural histories of several types of otosclerosis. Pathologic variations have been described. Guild [7] found that the otosclerotic focus may be located at any point on the labyrinthine capsule but that the area of greatest predilection is the anterior margin of the oval window. Nylen [8] investigated 121 temporal bones showing otosclerotic plaques. A single focus was present in 65% of the cases, while the remainder had two or more foci. The process was localized at the region of the oval window, with ankylosis of the stapes in 50%; at the area of the round window, 40%; at the cochlear capsule, 35%, and at the semicircular canals, 15%. Diffuse otosclerosis was present in 10% of the cases, the specimens of which often showed compression of the lumina of the cochlea and semicircular canals. While performing the fenestration operation, Seligman and Shambaugh [9] observed the presence of otosclerotic bone in the osseous horizontal semicircular canal in 2.9% of a series of 2,086 cases. These authors found the incidence of nerve degeneration to be more than twice as great in the patients showing a focus of otosclerosis in the semicircular canal than in those without this complication. It is obvious, therefore, that the location of the lesion must have a profound influence on the pathologic physiology. An

otosclerotic mass pressing on the semicircular canal would be a constant irritant, and the symptomatology of irritative labyrinthitis might be presented by the patient. When an otosclerotic new formation encroaches upon the oval window, which is already largely filled by the foot plate of the stapes, membrane, and ligament, a conglomerate mass forms and the joint becomes ankylosed. All sound waves coming by way of the ossicles will meet an obstructing body at the oval window, and a greater amount of energy will be necessary for the sound waves to penetrate into the scala vestibuli and mobilize the labyrinthine fluids. On the other hand, when the round window is involved, the high frequencies will be more adversely affected if there is no concomitant ankylosis of the foot plate of the stapes. Brunner [5] states that the pathology of otosclerosis includes the frequent occurrence of neural atrophy of the basilar portion of the cochlea. What impressed me most in otosclerosis was the functional viability of the part of the cochlea not affected by atrophy. In Case 2, fair rehabilitation was possible in the presence of the meagerest residual hearing. In Case 3, the beneficial effect of the fenestration operation was noteworthy (in spite of the almost complete ablation of the upper half of the auditory range). Brunner [5] considers the otosclerotic lesion a true neoplasm of the labyrinthine capsule. As such, it must be a tumor with a delineated deleterious effect, leaving the rest of the organ perfectly intact.

In a recent text, Cawthorne [10] states that the chance of hearing being improved by the fenestration operation in the unfavorable group of otosclerotics is about 50%; in the borderline group, 70%; in the favorable group, 80%. The difference in the prospect between the patient with advanced neural degeneration and the one with a mere obstruction is rather slight. It must be that in the "unfavorable group," no widespread destruction of nerve tissue takes place. Being a circumscribed tumor, the otosclerotic plaque has a limited deleterious effect, constituting an obstructive barrier in most cases, while resulting in basal atrophy in some instances. The unique rehabilitative capacity justifies the assumption that the uninvolved tissue of the inner ear retains its functional viability to the full extent in otosclerosis.

The majority of the patients with otosclerosis who come to the Temple Hearing Clinic for consultation are not ideal candidates for the fenestration operation. At the Mayo Clinic, Williams [11] found that one in three prospects belonged to Class A. Some are too old; others have physical or psychological contraindications to surgery; many show a perceptive component by their audiometric and tuning-fork responses.

It appears to me that the fenestration operation may be offered with greater confidence to otosclerotics with "some neural degeneration." The operation, as it creates a new physiologic mechanism of hearing, gives, in the majority of these cases, some improvement in hearing and considerable amelioration from tinnitus. The obvious contraindications include advanced age, poor state of health, complicating irritative labyrinthitis, and total or subtotal deafness of the other ear.

1923 Spruce St.

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MODIFIED TECHNIQUE FOR FENESTRATION OPERATION

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A FELLOWSHIP at Northwestern University in fenestration surgery under the supervision of Dr. Shambaugh was granted to one of us (Francisco) in 1947. When he returned to Spain the "Northwestern improved technique" was applied to approximately 100 fenestrations, with about 80% good results.

The success of the fenestration operation depends on the correct selection of cases and an exacting surgical technique to prevent osteogenic closure and postoperative serous labyrinthitis. Shambaugh's description of the ideal fenestra as one that is made "microimmaculate, enchondralized, periosteum-epidermis gloved, domed and burnished, with perfect asepsis and hemostasis" was a step forward toward complete success. Recently Lempert, after revising 100 fenestrations, advised invaginating the tympanomeatal flap into the fenestral gap and placing a small piece of cotton against it to facilitate healing of the flap with the fenestral margin to prevent osteogenesis.

In order to combine these ideas of two of the chief contributors to the development of the fenestration operation, we have made two modifications in technique.

The first consists in creating a step-like window with the superior edge higher than the inferior one. This is done by first creating the window in the usual site, with the edges level with the endolymphatic labyrinth. Then the edge of the fenestra toward the facial nerve is taken down a few tenths of a millimeter with a diamond burr or curette. The binocular operating microscope facilitates this procedure. The tympanomeatal flap now lies against this fenestra like a carpet on a staircase, insuring a close approximation with the endolymphatic labyrinth and contact with the endosteum at the fenestral edges (Fig. 1).

The second modification was adopted after incidents encountered during fenestrations and experience gained in skin grafting radical mastoidectomies since 1948. It had been observed that this graft always took, whether placed directly on the bone or bridged across the gap between the tympanic membrane and attic, in the modified radical operation.

Then in one fenestration the flap was thick and edematous, with persistent bleeding. For fear of labyrinthitis, the fenestra was not made, but the flap was cut free from the tympanic membrane, thinned, and replaced as a free graft over the semicircular canal and in contact with the upper edge of the tympanic membrane. The graft healed well, and in a second stage the fenestra was made. This same

experience was repeated in two more cases, but the fourth time that a thick edematous bleeding flap was encountered, the operation was completed in one stage, the fenestra being covered by the flap cut off as a free graft, with a good result.

Toward the end of 1950, after the labyrinth had already been opened during a fenestration operation, the burr accidently caught and completely destroyed the tympanomeatal flap. As the only resort a free split-thickness skin graft from the patient's thigh was used to cover the fenestra. As the result in this case was excellent, henceforth whenever the plastic flap was not entirely satisfactory, a free split-thickness graft was used to cover the fenestra. Later the free graft technique was adopted routinely.

The modified technique for the fenestration operation that we now use is as follows:

General anesthesia is administered, with controlled hypotension.

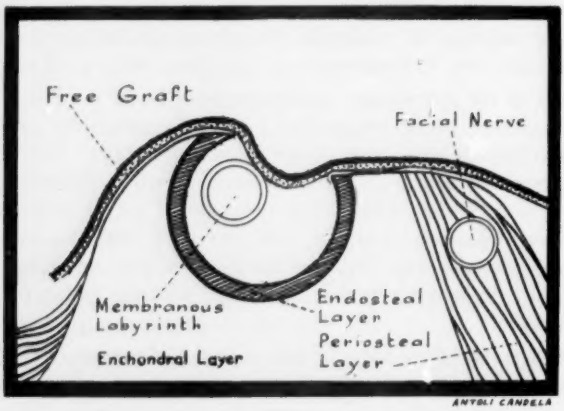

Fig. 1.—The step-like window, with the facial edge at a lower level than the upper edge.

Shambaugh's modification of the Lempert endaural incision is employed to expose the mastoid.

The skin of the meatus is cut by two incisions: the first along the posterior-inferior meatal wall to within 1 mm. of the tympanic membrane; the second beginning at the inner tympanic extremity of the first incision and extending superiorly about 1 mm. above Sharpnell's membrane to 12 o'clock (Fig. 2). There are thus two flaps—one that lies against the anterior wall of the cavity and the other that lies posteriorly over the facial ridge.

The fenestra in the semicircular canal is then made according to Lempert's technique with Shambaugh's modifications. The fenestra is covered by a free split-thickness graft extending to the edge of the tympanic membrane (Fig. 3). Shambaugh's sponge packs are then used.

Postoperatively, 800,000 units of penicillin and an ampule of an antihistamine drug are injected daily for five days. A laxative is given on the third day. The first dressing is done on the fifth day, the sponges being removed. The second dressing

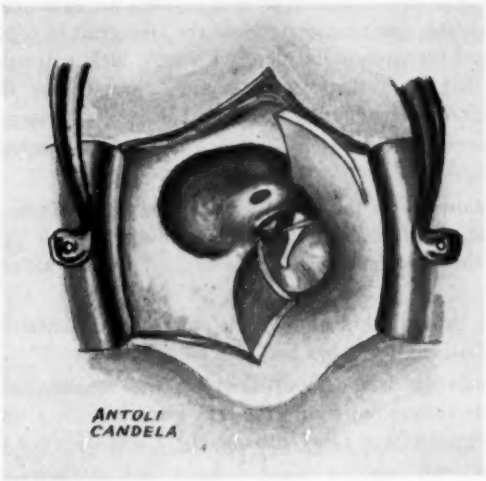

Fig. 2.—The skin of the meatus, with the two flaps made by the incisions.

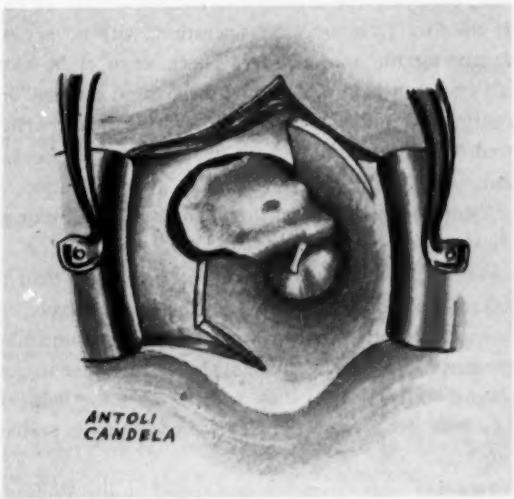

Fig. 3.—The fenestra covered by a free-thickness graft extending to the edge of the tympanic membrane.

is done on the 10th day, and the small gauze packs placed against the graft are removed. The third dressing is done on the 18th to the 20th day, loose desquamated epidermis being removed from the outer surface of the graft.

In our early operations a large graft that covered the entire cavity was used, but considerable exudation was observed. Now the free graft is reduced to almost the size of the classical tympanomeatal flap, with very little exudation and rapid epidermization, so that no further dressings are required after three to six weeks from the date of fenestration.

From our observations the step window and free graft technique possesses the following advantages:

(*a*) The window "faces the sound" more directly, facilitating the reception of sound waves; the thin free graft more easily invaginates to produce an intimate connection with the endosteum of the fenestral edges and with the endolymphatic labyrinth.

(*b*) The free graft is thin and pliable, so that the fenestra is clearly visible through it immediately after it has been placed.

(*c*) As Shambaugh has demonstrated on the monkey, stratified squamous epithelium inhibits osteogenesis, and the free graft is such a tissue. Moreover, it does not bleed, does not have adherent bone chips, and does not show postoperative inflammatory reactions.

The postoperative course in these cases was marked by very little labyrinthine reaction. Of 56 patients, only 2 had nausea and vomiting, with nystagmus toward the unoperated ear, requiring the injection of sucrose. Only 11 had vomiting three or four times for the first 12 hours after operation, with a very weak spontaneous nystagmus, disappearing the second day. There were eight more cases without vomiting but with a weak nystagmus toward the treated ear that disappeared on the third day. Two patients vomited on the third day after the cathartic. The remaining 33 patients showed no nausea, vomiting, or nystagmus.

Fifteen patients were out of bed the first day after operation, 39 on the second day, and most of them were walking on the third day, but all experienced a sense of instability that disappeared in 2 to 12 weeks.

The above-mentioned vestibular symptoms were far less than those observed in the preceding 100 fenestrations, in which vomiting was the rule, with some vertigo and nystagmus toward the untreated ear, for which intravenous injections of sucrose were given frequently. These earlier patients were reluctant to get out of bed, and only on the fifth and sixth day did they walk, with some help. Nevertheless, the sense of instability lasts longer with the free graft technique, perhaps because of the thinness of the graft.

The cochlear function after operation was tested by the Weber test, which localized to the treated ear in 51 patients who received good hearing results. There were 5 failures in the 56 operations; 3 of these patients also localized the Weber test to the operated ear, one did not localize to either ear, and one localized 512 and 256 frequencies to the treated ear and 1027 frequency to the untreated ear. Of these five failures, four were believed to be due to improper selection, with insufficient cochlear reserve, and one was believed to be due to a technical error.

MODIFIED FENESTRATION TECHNIQUE

Shambaugh states that "the earlier the hearing is improved after operation, the less the cochlear damage from labyrinthitis." Subjectively, the patients operated on by the modified technique all heard better from the first postoperative day, with the exception of the five with the unsuccessful operations.

Four patients who had had one ear operated on by the classical tympanomeatal flap technique were operated on in the second ear by the modified technique. All

TABLE 1.—*Distribution of 41 Patients Operated on by the Modified Fenestration Technique, Showing Results Obtained 15 Days After Operation*

Type	Total No.	Unimproved, No.	Decibels Gained*						Improved, Total No.
			10-15	16-20	21-25	26-30	31-40	41-45	
A.....	21	1	2	6	7	1	3	1	20
B.....	11	..	1	3	3	3	1	..	11
C.....	9	1	3	2	..	1	2	..	8
Total.....	41	2	6	11	10	5	6	1	39
%.....	..	4.8	14.6	26.8	24.3	12.1	14.6	2.4	95.1

* The total average gain was 21.07 + db.

TABLE 2.—*Distribution of 41 Patients Operated on by the Standard Tympanomeatal Flap Technique, Showing Results Obtained 15 Days After Operation*

Type	Total No.	Unimproved, No.	Decibels Gained*						Improved, Total No.
			10-15	16-20	21-25	26-30	31-40	41-45	
A.....	21	2	10	8	1	..	..	..	19
B.....	11	2	6	3	..	..	..	..	9
C.....	9	4	5	..	..	..	..	..	5
Total.....	41	8	21	11	1	..	..	..	33
%.....	..	19.5	51.2	26.8	2.4	..	..	..	80.4

* The total average gain was 10.90 + db.

TABLE 3.—*Comparison of the Results Obtained from the Two Types of Fenestration Techniques*

Fenestration Technique, Type	Patients, No.	Average Amount Gained, Db.*
Modified free graft.....	41	21.07 +
Standard tympanomeatal flap.....	41	10.90 +

* The two-week tests were made at frequencies of 512, 1024, and 2048.

agreed that the postoperative course was much easier and that the hearing improvement appeared earlier. This was confirmed audiometrically.

An audiogram was made 15 days after operation for 41 of the patients, all of whom experienced a good result. In this first test after operation an improvement for the low and speech frequencies was observed in 39 patients, while the high frequencies were unchanged or a little worse.

Tables 1 and 2 show the improvement in decibels at the first postoperative audiogram 15 days after operation in 41 cases operated by the standard tympanomeatal flap technique and in 41 cases operated by the modified free graft technique.

Summing up these observations we may conclude that the postoperative serous labyrinthitis is definitely less with the step window and free skin graft technique than with the usual technique previously performed by us (Fig. 3).

So far the 56 cases operated on by the modified technique have shown no evidence of osteogenic closure, but it is too early to draw conclusions. In 19 cases one

TABLE 4.—*Hearing Results Obtained at Least One Year After 19 Patients Were Operated on by the Standard Fenestration Technique*

	Patients					
Type	Total No.	Unimproved, No.	Gained Between 15 and 60 Decibels, No.	Reached Practical Level, No.	Improved, Total No.	Average Amount Gained, Db.*†
A	6	..	3	3	6	30.3 +
B	9	1	7	1	8	19.8 +
C	4	2	2	..	2	13.7 +
%	..	15.7	63.1	21.0	84.2	

* Tests were made at frequencies of 512, 1024, and 2048.
† The total average gain was 21.8 + db.

TABLE 5.—*Hearing Results Obtained at Least One Year After 19 Patients Were Operated on by the Modified Fenestration Technique*

	Patients					
Type	Total No.	Unimproved, No.	Gained Between 15 and 60 Decibels, No.	Reached Practical Level, No.	Improved, Total No.	Average Amount Gained, Db.*†
A	6	..	..	6	6	37.5 +
B	9	..	6	3	9	33.8 +
C	4	1	2	1	3	25.0 +
%	..	5.2	42.1	52.6	94.7	

* Tests were made at frequencies of 512, 1024, and 2048.
† The total average gain was 31.3 + db.

TABLE 6.—*Comparison of the Results Obtained from Tests Made at Least One Year After 19 Patients Were Operated on by the Standard Fenestration Technique and an Equal Number of Patients Were Operated on by the Modified Fenestration Technique*

	Patients		
Fenestration Technique, Type	Improved, %	Reached Practical Level, %	Average Amount Gained, Db.*
Modified	94.7	52.6	31.3 +
Standard	84.2	21.0	21.8 +

* Tests were made at frequencies of 512, 1024, and 2048.

to two years have elapsed since surgery, and all have maintained their improvement. In Tables 4, 5, and 6 the most recent hearing results in these 19 cases are compared with a similar series operated by the usual technique.

Figure 4 shows the hearing results of three patients (one of Class B and two of the early Class A) operated on by modified technique. Figure 5 shows the results of three patients of Class C suitably operated on by this technique.

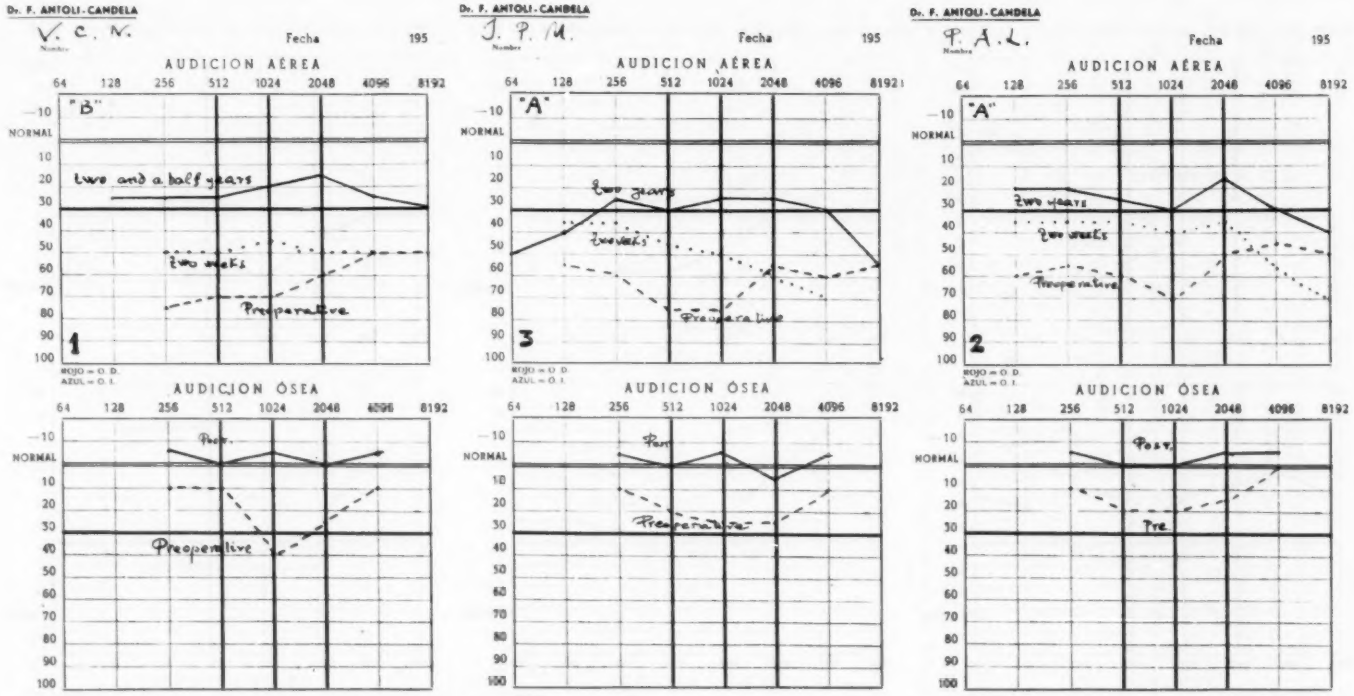

Fig. 4.—Audiograms obtained two years after three patients (one of Class B and two of Class A) were operated on by the modified fenestration technique. Note the good improvement by air and bone conduction.

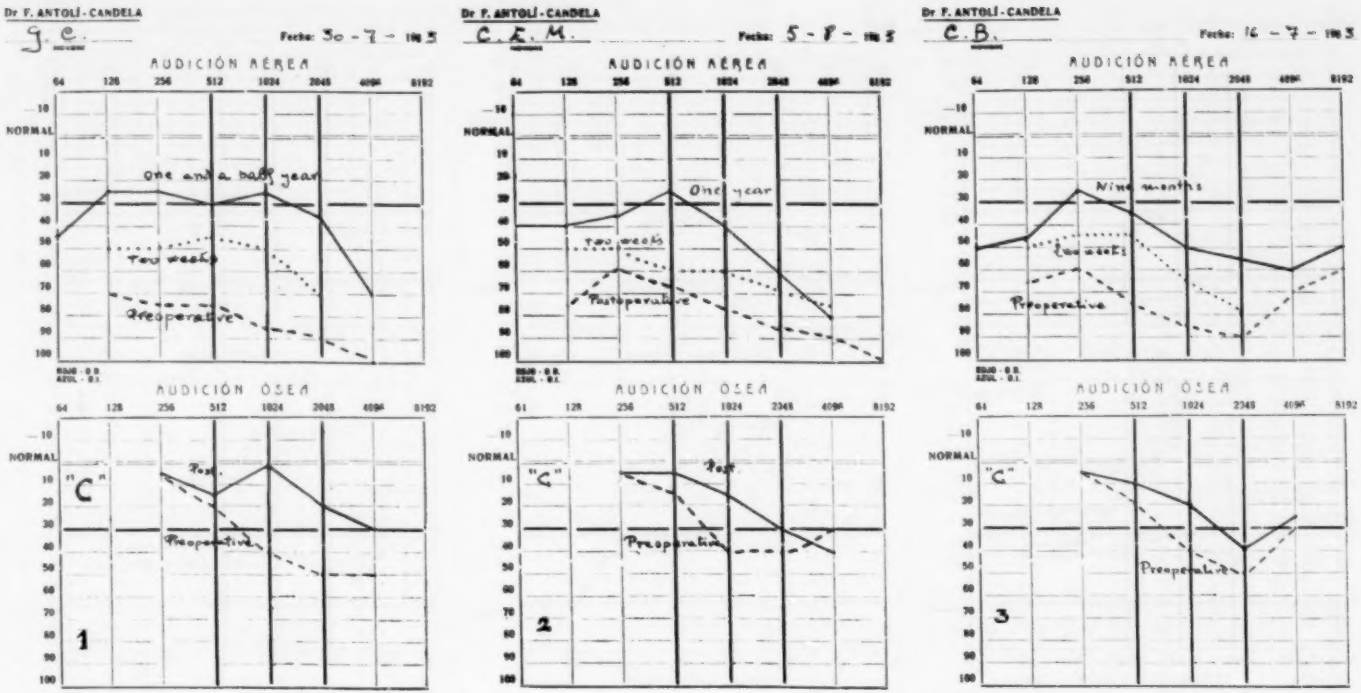

Fig. 5.—Audiograms of three patients of Class C who were operated on by the modified fenestration technique. Good improvement by air and bone conduction is shown.

SUMMARY AND CONCLUSIONS

A modified technique for fenestration of patients with otosclerosis is presented, consisting of a step-like window covered by a free split-thickness skin graft. The early hearing results as well as the results observed later suggest that this technique not only helps to prevent osteogenic closure but also especially prevents postoperative serous labyrinthitis provoked by the inflammatory reaction of the tympanomeatal flap. These results are substantially better than the results obtained in cases previously operated on by us by the usual technique. Further observations on a larger series followed for a longer time will be made.

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Case Reports

EPISTAXIS CONTROLLED BY COMBINED LIGATION OF THE EXTERNAL CAROTID AND ANTERIOR ETHMOIDAL ARTERIES

Report of a Case of Severe Epistaxis Occurring Seven Days Following Head Injury

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AND
HARRY W. FISHLER, M.D.
LOS ANGELES

DUAL LIGATION of the external carotid and anterior ethmoidal arteries in the initial surgical procedure for severe uncontrolled epistaxis has been performed numerous times, but scant mention of this combined operation appears in the literature. Oppenheim and his associates, [1] in 1952, in a comprehensive review of the literature at their disposal recorded 17 cases (including 2 of their own) in which the anterior ethmoidal artery was surgically interrupted either primary or secondary to the external, internal, and/or common carotid arteries, and in only one instance, that of Williams [2] in 1945, were the external carotid and anterior ethmoidal arteries ligated in one surgical session.

In 5 of the 17 cases recorded, ligation of the anterior ethmoidal artery alone stopped the hemorrhage. In eight cases (excluding that of Williams [2]) interruptions of the anterior ethmoidal artery were preceded by ligation of the external carotid artery at separate sessions, an interval of several hours to one or more days ensuing between operations. In the remaining cases, the common carotid and/or the internal carotid artery was ligated in conjunction with the anterior ethmoidal artery or as a secondary procedure.

The vascular anatomy of the nose has been described in detail by Weddell and his associates [3] (1946), Hallberg [4] (1950), and others, but for all practical purposes for this paper, the blood supply of the nose is derived from the branches of the external carotid artery via the internal maxillary and sphenopalatine vessels as well as from the internal carotid by way of the ophthalmic artery and its branches, namely, the anterior and posterior ethmoidal vessels. The posterior ethmoidal arteries are usually small in caliber and seldom a factor in ethmoidal epistaxis.

Surgical interruption of a major artery supplying the nose is resorted to in the presence of persistent epistaxis only after the usual conservative measures have failed. Since the sphenopalatine vessels supply the major part of the nose, it is logical to assume that in the case of severe uncontrolled epistaxis in which it is impossible to establish the bleeding point after careful suction and inspection ligation of the external carotid artery is the procedure of choice. However, if the bleeding still persists after all packings have been removed, interruption of the anterior ethmoidal artery usually controls the hemorrhage. Macbeth,[5] 1948, in referring to previous authors who suggested that failure of carotid ligation to control nasal

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bleeding was due to anastomosis of vessels across the midline, refutes this statement by noting, "In our experience if carotid ligation fails the reason is that the wrong arterial system has been interrupted, and in such cases ethmoidal interruption succeeds."

Interruption of the posterior ethmoidal artery by diathermy or section along with the anterior ethmoidal artery appears in four cases on record, but in each case the vessel was small and considered of little consequence. Ligation of the common or internal carotid arteries, according to Schorstein [6] in 1940, not only may cause serious cerebral damage in the presence of anemia due to hemorrhage but in the majority of cases is unsuccessful in the control of epistaxis from the anterior ethmoidal arteries. The free anastomosis between the internal carotid arteries at the base of the brain maintained through the circle of Willis is very efficient from the clinical and anatomical point of view.

Head injuries, no matter how trivial, play an important role in the etiology of severe epistaxis from the anterior ethmoidal arteries. Macbeth,[5] in reporting four cases of head injuries, emphasized the relationship of these injuries to anterior ethmoidal artery hemorrhage so concisely that, in our opinion, it bears repeating verbatim:

> In cases of head injury there is often a fracture through the ethmoid, or through the fronto-ethmoidal suture, and this may involve the anterior ethmoidal artery or one of its main branches. These vessels lie in bony canals where they are subject to mural laceration or erosion, and where consequently they are unable to contract and retract. Thus, although they are quite small, the hemorrhage to which they may give rise is often considerable.
>
> In these cases bleeding may be immediate or at an interval after the original accident—i. e., it may be secondary in type, and presumably initiated by infection. Four of our cases occurred after injuries; in three of them carotid ligation had failed to stop the bleeding, and ethmoidal ligation succeeded. In the fourth this latter operation was undertaken with success as the primary and only operative measure.

Williams [2] also reported a case of head injury in which the epistaxis was controlled only after the anterior ethmoidal artery was ligated secondary to the ligation of the external carotid artery.

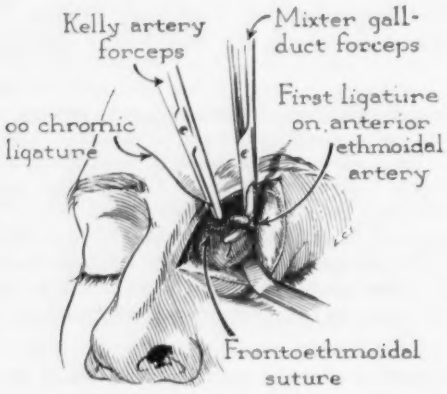
REPORT OF CASE

A. W., a 58-year-old white man, gave the following history: On the morning of Aug. 8, 1953, while at work shoveling lemons, he picked up a shovelful and as he raised up struck his head (the left occipital area) a sharp blow against a metal chute, causing him to "black out" momentarily. He grasped a nearby rail for support, and he was immediately aware of a sharp throbbing pain in his head in the region across the vertex. He reported the accident to the authorities and then continued to work in spite of this pain, which persisted unabated day and night.

One week subsequent to the accident, he bled a little from the left nostril before going to work. After he had worked for one and a half hours, the pain became so unbearable that he reported to his foreman, who advised him to go home and visit the doctor. The doctor was contacted in the Corona Hospital about 8 p. m., and he gave the patient some capsules and advised him to take one and go to bed immediately. About 9:30 p. m. that night, while undressing to retire, the patient had just bent over to remove the remaining sock when blood gushed from the left nostril. He was immediately transferred to the Corona Hospital, where he bled through the nose intermittently all through the night. This continued for two days, and on the third day he bled not only through the nose but from the mouth as well. This bleeding continued to get worse in spite of injections and two blood transfusions. The pain seemed to disappear with the onset of the bleeding and would return when the bleeding ceased throughout the entire episode.

On the sixth day, Dr. Leo Adelstein, the neurosurgeon consultant for the State Industrial Insurance Fund, had the patient transferred to the Queen of Angels Hospital in Los Angeles. He called one of us (Peluse) in consultation to treat the patient. On arrival at the hospital the patient was found sitting up in bed coughing and spitting blood from a profuse epistaxis that gushed from both nostrils and his mouth into an emesis basin almost filled with blood. With the aid of suction it was possible to determine that the bleeding was from the left naris posteriorly. A tight posterior and anterior nasal pack was inserted, which appeared to check the bleeding. Sedation was ordered, but several times throughout the night he bled through the packs.

The following morning the patient was found sitting up in bed with blood still oozing through the anterior pack and into the pharynx. He was very despondent and complained bitterly of the pain in the throat when he swallowed. He refused to swallow even liquids and was miserable in general. His pulse was somewhat irregular, and he was slightly cyanotic due to his inability to cough up the clotted blood in his hypopharynx. The throat was cleansed with suction and the strings of the posterior pack tightened, and this seemed to check the oozing. At noon, we visited the patient, and one of us (Fishler) removed the bloody packs and reinserted the posterior and a tight anterior nasal pack. The bleeding was checked for a short time, but blood



Schematic representation of the anterior ethmoidal artery in the orbit and our improvised method of ligature application. (Anatomically the anterior ethmoidal artery in the orbit is surrounded by a cuff of periorbital connective tissue and is accompanied by the anterior ethmoidal vein, the ethmoidal branch of the nasociliary nerve, and the lymphatics. This structure is relatively large.)

began to ooze through both packs and the oozing continued throughout the rest of the day and night. The patient was given 500 cc. of whole blood intravenously.

We visited the patient again the next morning and found his condition critical. The cyanosis was deeper; the breathing and pulse were irregular. He appeared stuporous and could be aroused, but he would immediately fall back into a muttering delirium. He was ordered to surgery.

First Surgical Procedure.—With the patient under intratracheal anesthesia, an incision was made along the anterior border of the left sternocleidomastoid muscle at the level of the thyroid cartilage. The platysma was separated and the carotid sheath opened with blunt dissection, exposing the common carotid artery. The bifurcation was located at the level of the angle of the jaw. The superior thyroid artery, which is usually found in the neck, was entirely absent. The first two branches of the external carotid artery were located at the angle of the jaw, one emerging anteriorly and the other posteriorly, at approximately the same level just cephalad to the bifurcation. Two nonabsorbable (silk) ligatures were applied between these branches and the bifurcation. The subcutaneous tissues and the skin were approximated and the wound dressed. The posterior and anterior nasal packs were removed and the left naris inspected. The blood

welled up alarmingly and seemed to be even more profuse than before surgery. The left orbit was prepared for exposure of the anterior ethmoidal artery first and, if necessary, the posterior ethmoidal artery subsequently.

Second Surgical Procedure. A left medial periorbital incision was made and the periosteum elevated from the orbital wall until the anterior ethmoidal artery with its cuff of connective tissue was identified proceeding to the anterior ethmoidal foramen. The orbital tissues were gently teased from the orbital wall in the vicinity of the anterior ethmoidal artery and retracted with a thin malleable ribbon retractor which had been fashioned to fit the orbit, aiding materially in a good exposure of the vessel. With the usual curved forceps and the other regular surgical instruments at hand on the operating table, we were unable to find a suitable ligature carrier to circumvent the anterior ethmoidal artery deep in the orbital recess; the surgical supervisor suggested use of a 9-in. (23-cm.) Mixter gall-duct forceps. Carefully, but with little difficulty, the jaws were passed under the vessel and then opened slightly. A chromic absorbable (gut) suture, No. 00, was threaded through the opened jaw with the aid of a Kelly mosquito forceps (Figure). Two ligatures were applied to the artery and then loose dressings laid over the open wound preparatory to the inspection of the nose. The nose was uncovered and suction applied to the left naris. The bleeding had stopped completely. The wound was then closed, dressings applied, and the patient returned to his ward with no packs in his nose. Two pints (950 cc.) of blood had been given to him in the operating room.

The postoperative course was stormy the first night, necessitating a special nurse, but subsequently it was uneventful, and the patient left the hospital in a week. X-rays of the skull and an encephalogram taken on the second postoperative day were negative. He complained of diplopia on the first postoperative day. This continued for approximately three weeks but finally resolved. His headaches returned immediately after the bleeding was controlled, but by the end of the fourth week postoperatively they had subsided. The patient was able to return to work in six weeks. Both wounds healed by first intention, and up to the date of writing the bleeding never recurred.

COMMENT

The role of head injuries as an etiologic factor in anterior ethmoidal artery hemorrhage should be given serious consideration. In each of the five cases of head injury with epistaxis reported, the bleeding was not controlled until the anterior ethmoidal artery was interrupted, either as a primary or as secondary surgical procedure. In this case, it could be surmised that the head injury, by contrecoup, had involved the anterior ethmoidal artery or one of its branches in their bony canals. The bleeding, which was of the secondary type, could have been initiated by infection at an interval after the original accident (in this case, seven days), as postulated by Macbeth.[5] The bleeding in this case, as in all the other accident cases reported, was not arrested until the anterior ethmoidal artery was ligated. From the knowledge gained from this case and from Macbeth's [5] and Williams' [2] experiences, it may be well to keep in mind the interruption of the anterior ethmoidal artery as the initial procedure in severe epistaxis following head injuries.

The Ferris-Smith set of anterior ethmoidal artery instruments, namely, the ligature carrier, knot tier, short and long orbital retractors, is available in many of the well-equipped otolaryngological operating rooms. The "gall-duct forceps" is standard equipment in most of the general surgery operating rooms, and it proved to us a satisfactory improvisation and facilitated the ligation of the anterior ethmoidal artery immensely. The one provided us was the regular 9-in. Mixter gall-duct forceps. Silver clips, as used by neurosurgeons for interruptions of blood vessels, are practicable in this procedure. They have been used many times in the cases reported, but we have had no practical experience with them and cannot comment on their use.

Intratracheal anesthesia, as used in this case, is, in our opinion, the one of choice. The patient is under the constant control of the anesthetist, who can keep the hypopharynx cleared of blood with suction. Since the larynx is blocked off, there is little chance for blood to seep into the trachea. There is also less shock to the patient, especially if the surgical procedures are involved and prolonged, as occurred in this case.

Ligation of the external carotid artery was the first surgical procedure in the plan we had evolved previous to surgery to be followed by ligation of the anterior ethmoidal artery if the bleeding had not stopped. We were prepared to follow through with the interruption of the posterior ethmoidal artery, but this proved unnecessary. The added hazard of the close proximity of the optic nerve must be taken into consideration when interruption of the posterior ethmoidal artery is contemplated.

All the packs were removed from the nose on the operating table and sufficient time allowed to be absolutely sure the bleeding was controlled. Because of the critical condition of this patient at the time of surgery, any further surgical procedure subsequently could have resulted disastrously.

It is worthy to note that although the bleeding had started seven days after the head injury, the State Industrial Insurance Fund did accept this case as fully compensable.

SUMMARY AND CONCLUSIONS

A case of head injury, followed in seven days by severe epistaxis, is reported in which the combined ligation of the external carotid artery followed in sequence by ligation of the anterior ethmoidal artery in the initial surgical procedure controlled the hemorrhage and proved to be a lifesaving measure.

It is our conjecture that the accidental striking of the left occipital area against a metal chute could, by contrecoup, have involved the left anterior ethmoidal artery and by secondary infection and erosion precipitated the severe epistaxis seven days later.

Ligation of the anterior ethmoidal artery as the initial surgical procedure in cases of severe epistaxis following head injuries is the object lesson deduced from the experience of the authors reporting these types of cases as well as in this particular case.

An anomalous left external carotid artery was encountered in which the superior thyroid artery, which usually is found in the neck, was entirely absent, and the bifurcation was located high up at the level of the angle of the jaw.

Intratracheal anesthesia, in our opinion, was the ideal choice of anesthetic in this case.

No special instruments were needed for the ligation of the anterior ethmoidal artery. A thin malleable ribbon retractor, fashioned to retract the orbital tissues for the exposure of the anterior ethmoidal artery in the orbit, and a 9-in. (23-cm.) Mixter gall-duct forceps to circumvent the vessel proved to be suitable improvisations to facilitate the ligation.

The nasal packs were removed on the operating table after the first surgical arterial interruption (external carotid artery). Since the bleeding persisted, the second major arterial branch supplying the nose (anterior ethmoidal artery) was interrupted, following which the bleeding ceased immediately.

This case was acceptable as fully compensable by the State Industrial Fund, without any reservations.

1930 Wilshire Blvd. (57).

Sister Carolita, surgical supervisor, suggested the use of the gall-duct forceps.

Mrs. Lucille Cassell Innes, of the Department of Visual Education, College of Medical Evangelists, supplied the illustration.

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Progress in Otolaryngology

AUDIOLOGY

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1950, 1951, and 1952

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FOR THE purpose of this review the term audiology is considered in its broadest concept to include all aspects of hearing. The scope of the subject precludes a comprehensive consideration of all articles. Excellent reviews related to hearing have already been presented by Lewy,[1] Juers,[2] Shuster,[3] and their colleagues in the A. M. A. ARCHIVES OF OTOLARYNGOLOGY. The present review is intended to supplement these and will accordingly avoid references to otosclerosis, fenestration, the results of therapy in certain hearing disorders and noise, acoustic trauma, and industrial deafness. Since our object is to integrate rather than delineate the many facets of audiology, subheadings are avoided. Instead this review will have a tendency to progress from purely clinical to essentially basic material, with more emphasis on the latter. To facilitate its use, the following index is included:

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From the Department of Otology, Jefferson Medical College.

Walsh [4] states that audiology has to do with measuring and managing hearing impairment and that it comprises an area rather than a finite discipline. A university audiology center has three functions: (1) teaching, (2) research, and (3) service to the deaf and hard of hearing. Walsh stresses the important role which the otologist must play in all these functions, particularly the last. He feels that every patient going to an audiologic clinic should have a thorough otologic study, and that the otologist must be acquainted with all aspects for the proper management of the deaf and hard of hearing patient. The otologist should be captain of the team which renders clinical service to the patient.

Silverman,[5] in his introductory remarks in a "Symposium on Audiology," discusses the scope of audiology without actually defining it. Some of the personnel associated in audiology include speech correctionists, teachers of the deaf, otologists, acoustic physicists, clinical and experimental psychologists, physiologists, public health experts, and others.

One of the most comprehensive perspectives of audiology and its relation to the otologist is presented by Lederer and Marcus.[11] With the enthusiasm of pioneers in this newly appreciated field, the authors describe the numerous facets of audiology, including physiology, pathology, acoustic physics, psychology, electronics, speech pathology, and education. They urge the otologist to assume with a team the complete responsibility for the hard of hearing patient as a whole entity, and point out the direction for future advances in various phases of audiology.

Hoople,[10] an outstanding proponent of audiology, emphasizes the importance of rehabilitation and the more active role otologists should assume in audiological work. He urges the establishment of more hearing centers by otologists in conjunction with the community. He describes the great value of speech audiometry and its usefulness in testing young children. The reliability of the psychogalvanic skin resistance test in certain cases is established, particularly in testing the preschool child and in psychogenic deafness. Hoople crystallizes the importance and methods of auditory training and states that it is essential for those with irreversible hearing losses, whether or not a hearing aid is worn.

Hill [12] presents a most constructive and practical program for auditory rehabilitation with little funds. He emphasizes that what is vital is service to the hard of hearing child, not so many facilities. The essential elements for a satisfactory program are an interested otologist and a competent teacher for hard of hearing children. Volunteer workers can be obtained, in addition, by good public education. Hill describes his most impressive clinic in Waterville, Maine, where 31 youngsters and their parents from all over the state are guided and helped. The children attend their local schools and get supplementary work in speech and hearing. Equipment was obtained long after the clinic was functioning under the excellent leadership of a competent teacher. Hill clearly demonstrates that service can be rendered to these children on a so-called "shoe string" if the motivation and interest of the otologist are great enough.

Reger,[6] in discussing the minimal requirements of equipment for audiological work, stresses that equipment is a means to an end and not an end in itself. He states that "unless the operation and limitations of the equipment are known and observed, more and bigger mistakes will be made with equipment than without it." The most important instrument is a speech audiometer, and he explains how useful this is

in plotting a speech-function curve and in diagnosis. It is also useful in convincing a parent that a child has a hearing loss. The next important instruments, according to Reger, are a pure-tone audiometer and tuning forks. A few noise makers or percussion toys are also necessary when working with children. As far as research equipment is concerned, it should be limited only by lack of imagination or lack of funds. The training and scientific integrity of the person who uses the equipment is of utmost importance.

A significant contribution is Bergman's [13] manual for planning an aural rehabilitation clinic. The manual contains details concerning instructional programs, sound rooms, design, equipment cost, and personnel requirements. Despite many changes occurring since 1950, the manual still has much sound information for those planning hearing centers.

Lederer and Marcus [14] stress the personal, economic, and social problems of the hard of hearing adult and encourage the otologist to help solve these problems. Intelligibility testing and evaluation of hearing aids are essential in the program along with treating the patient rather than his hearing handicap alone. They describe a course in auditory education preparatory to the actual selection of an aid. During this course the patient receives insight into his problems as well as auditory training and psychological adjustment.

Silverman,[15] one of the ablest investigators in clinical audiology, explains the underlying principles and technique of speech tests (articulation tests) and how they may be used in clinical situations. Basically, articulation testing expresses the relation between the percentage of words (or sentences) heard correctly and the intensity of the words at the ear of the listener. An articulation curve is formed by plotting on a graph the per cent of words heard against the intensity in 10-db. steps. Within certain limits the louder the speech, the more words are heard. He distinguishes three thresholds in articulation tests:

(*a*). Threshold of detectability, at which speech is heard about half the time but is not understood

(*b*). Threshold of perceptibility, at which the listener begins to perceive some words but cannot follow connected speech

(*c*). Threshold of intelligibility, which is the intensity level at which a listener understands half the speech presented to him without perceptible effort

For articulation testing, the last threshold is the important one. Many variables influence the results of this threshold. Some of these are the type of words or speech used, the speaker's voice, the distance from the microphone, the fidelity of the electronic equipment, and many others. To meet necessary criteria to determine thresholds of intelligibility, lists of 42 bisyllabic words of spondee stress were developed. These words are called spondee words, and all syllables are equally stressed.

Silverman emphasizes the need for not only testing the threshold of hearing but also determining how the ear hears speech above threshold, especially at normal speech levels. The ability to distinguish one sound from another when both are audible is called discrimination, as distinguished from sensitivity, which refers to the faintest sound which the ear can hear. To test discrimination, another list of words is used (to satisfy different criteria). This list is called the PB (phonetically

balanced) list. With these words, a different type of curve is plotted, using per cent of words correct against intensity levels. Using these two types of articulation tests properly, one can obtain considerable information regarding diagnosis, prognosis, and evaluating medical and surgical procedures and hearing aids. Silverman explains how these tests aid in selecting suitable patients for fenestration surgery. The different results in conductive and nerve deafness are described.

The evaluation and concept of social adequacy are very clearly explained and Silverman states, "the social adequacy index is essentially the average percentage of phonetically balanced words that would be correctly understood by the patient at the faint, the conversational and the loud speech levels." This social adequacy is advocated to evaluate the remedial measures in terms of shifts, both in sensitivity and discrimination. Silverman emphasizes that articulation tests are not intended to supplant other tests but to supplement them.

Hoople [16] indicates that speech audiometers possessing proper calibration and attenuation are being used to determine speech reception thresholds and discrimination scores. For testing thresholds, bisyllabic words, known as spondee words, are used. For determining discrimination scores, monosyllabic words, usually in lists of 50 words, are used and called PB, or phonetically balanced, words. The discrimination test is generally begun at 25 db. above the speech reception threshold. The score is the percentage of words heard correctly. If with increased intensity there is no improvement in the discrimination score, a perceptive loss is probably present. Testing with speech audiometry is rapidly becoming a standard test.

In a 125-page comprehensive treatise on "Finnish Speech Audiometry," Palva [17] stresses the importance of speech audiometry, especially in prescribing hearing aids and evaluating suitable fenestration patients. He advocates the use of speech audiometry for a better understanding of the degree and type of hearing loss present.

Weille [18] feels that the great practical value of speech audiometry is in checking the validity of air and bone audiometry. He describes his own speech audiometry and states that a sound-dead room is not necessary A room having a noise level of less than 50 db. is adequate. Weille reviews some basic facts in speech audiometry, defining spondee and PB words so that an otologist can see the practical usefulness. The spondee words are used to determine threshold of hearing, while PB lists are used for intelligibility tests above threshold. A well-constructed audiometer can also be used as a "master" hearing aid, helping the otologist to predict the usefulness of a hearing aid in specific cases.

Glorig * describes the measurement of hearing loss, stating that spondee words emphasize vowel sounds or frequencies from 300 to 1500 cps, whereas, PB, or phonetically balanced, words emphasize the consonant sounds from 1500 to 3000 cps. Speech tests are not generally accepted yet because of lack of adequate equipment and completely standardized material. Equipment for pure-tone testing includes an audiometer capable of making tones from 250 to 8000 cps with a controlled system of attenuation, a masking-noise device for measuring hearing where there is more than 30 db. difference between two ears, and a good earphone to transduce electrical energy to physical energy. Equipment for speech audiometry includes

* Glorig, A.: The Measurement of Hearing Loss, Third National Abatement Symposium, Armour Research Foundation of Illinois Institute of Technology, Chicago, Oct. 10, 1952.

microphone with preamplifier for input power, monitor to regulate input, attenuator to control output, speaker or earphones with output amplifier, and recorder for use with recorded testing. Standard testing conditions are required with less than a 40 db. ambient noise level. Better conditions are needed for testing at threshold for normal hearing than are needed for screening at above threshold.

One of the most lucid and clarifying discussions of hearing aids and how they work is presented by Hirsh.[7] He enumerates the essential parts of a hearing aid and distinguishes between amplification and attenuation as well as giving a very clear and concise account of the decibel. The importance of measuring not only threshold but above threshold characteristics is stressed. The former is determined with relatively simple material, such as sentences or spondee words; whereas, the latter is determined with words in phonetically balanced lists. Amplification alone is insufficient to improve the ability to discriminate. This is one reason for the difficulty nerve-deafened patients encounter in using a hearing aid.

Glorig [19] gives the function of a hearing aid as a modified amplifier which converts sound into electrical energy, amplifies it 100 million times, and reconverts it to physical energy. This process may introduce changes in frequency response, amplification distortion, and extraneous noise. He stresses that auditory training is important for patients with perception deafness. His experiences at an Army audiological center show that a highly selective procedure for hearing aids is not necessary for conductive losses and 80% of perceptive losses. An adequate training program is most essential. Glorig [20] also feels that the difficulties in obtaining adequate benefit from a hearing aid occur in patients with predominantly nerve loss. The average gain for speech with an aid is 40 to 45 db. Factors influencing the beneficial effect of an aid are the duration of deafness, effect of ambient noise, recruitment, age, and economic and social background of the patient. Auditory training is of utmost importance in satisfactory use of a hearing aid. Air-conduction ear pieces, individually fitted, should be used except in cases of active infection in the ear. The author describes a soft plastic ear insert which he developed, and which produces a nearly perfect acoutic seal and is comfortable.

The findings from 476 questionnaires sent by van Deventer and Scanlon [21] to industry revealed the following: (1) a man using a hearing aid is more likely to be hired than one who has a hearing loss and will not wear a hearing aid; (2) a hearing-aid user is more acceptable for employment than men with other disabilities; (3) business believes in the selective placement of the hearing-aid wearer.

The excellent results of and need for auditory training programs are manifested by Johnson and Siegenthaler,[22] who report changes in the behavior, attitudes, and skills of patients during the program. The authors outline 20 lessons for group auditory training. Included in the lessons are the care and use of the hearing aid, auditory discrimination tests, anatomy and physiology of the ear, and phonetics of speech.

Siegenthaler and Gunn [23] examine 143 case records of hearing-aid selections and find that 67% have perceptive losses, 25% mixed, and 8% conductive. All these patients, irrespective of the type of deafness, received improvement in threshold gain with their hearing aids. The conductive type of loss was, of course, helped much more than the others.

In a most impressive and comprehensive thesis, Morrissett [24] gives a critical analysis of present-day treatment of nerve deafness. Reports of excellent results in nerve deafness upon administering medication are shown to be unscientific and lacking controls and accurate objective testing. Some of these reports included the use of neostigmine, vitamins, benzyl cinnamate, and amino acid. In the author's vast personal experience with many advocated methods for curing nerve deafness, he states with sincerity and critical detachment, "Not a single patient with nerve deafness . . . showed any significant sustained improvement." Appreciating the futility of present medical treatment of nerve deafness, Morrissett presents an integrated program of aural rehabilitation in civilian life. He states that the program should be supervised by otologists but should include the psychologist, teacher of speech reading, sound engineer, speech teacher, and others.

Fletcher [25] feels that the validity of calculating hearing loss for speech from a pure-tone audiogram by averaging the loss in the frequencies 500, 1000, and 2000 has received much criticism. He suggests a new formula which uses the same frequencies but takes the average of the two smallest values of hearing loss. The formula is based on studies of loudness, including the relative contributions of different frequency regions to the audibility of speech at or near threshold level. When tested on 165 ears, the new formula seemed to give better results than the old one. The formula is particularly better when the audiogram is not "flat."

MacDonald [26] concludes from studies of roentgenograms and audiological findings that absorption of sound in the mastoid air cells is an important factor in determining bone conduction. He describes an office procedure for obviating this difficulty and determining the effect of air-cell absorption. The test, he concludes, is too time-consuming at present but warrants further investigation.

Carhart [27] reviews the major difficulties in bone-conduction testing and shows that, if properly done, such testing is very reliable and superior in repeatability to thresholds for air conduction. If the test is done with a hearing aid type bone receiver held in a steel headband, pressure and positioning are not significant variables. The values of bone-conduction testing, especially for diagnostic value, are presented.

Woods [28] presents the results of a 15-year study on bone conduction in otosclerosis. His results show an 11.4 db. average improvement in bone conduction in the operated on ear as compared with no change in the ear not operated on, which was used as a control. In all testing, a 70 to 80 db. masking noise produced by an electric buzzer was used. Ninety per cent of the cases showed improvement in bone conduction postoperatively. Woods feels that stapes fixation, per se, causes a loss in bone conduction and that otosclerosis is initially a mixed deafness.

Markle and his associates [29] state that the most usual criteria for the employment of masking are, for air conduction, a difference of 40 db. or more between the air-conduction thresholds and, for bone conduction, a 15 db. difference between the air-conduction thresholds or lateralization of the bone-conducted tone to the ear not being evaluated. Since effective masking can only be derived by probing, which is time-consuming, the authors find 85 db. of white or saw-tooth noise satisfactory for the majority of cases for air-conduction measurement. Exceptions to this will, of course, be encountered. In order to determine which ear to test first for bone conduction, they suggest using the bone receiver on the audiometer

for a Weber test. They present an excellent and logical description of their method and discuss overmasking and testing unilateral losses with the opposite ear almost normal. They suggest the following procedure for routine audiograms:

1. Air-conduction thresholds of the better ear
2. Air-conduction thresholds of the poorer ear, with 85 db. masking if there is a 40 db. or more difference
3. Weber test at 10 db. above the mid-line threshold
4. Weber test at maximum output of audiometer
5. Bone-conduction threshold of the lateralized ear or ears
6. Bone-conduction threshold of the nonlateralized ear, with 75 db. masking presented to the lateralized ear
7. Fowler loudness balance test, if indicated

Campbell [30] urges more application of the impedence formula in conduction deafness. The formula states that factors increasing stiffness in the conductive mechanism produce hearing loss predominantly in the lower frequencies, whereas increase in mass will cause loss predominantly in the higher frequencies. Campbell admits, however, that application of this formula is not always simple in the ear, with its many variable influences and complex mechanisms.

Fowler [31] points out that the term "sudden deafness" is vague and may mean actual sudden onset or a sudden awareness of a deafness (usually unilateral) that has been present for many months or years. The author discusses the findings in 26 personal cases of actual permanent sudden deafness and also 34 cases reported by other authors. In almost all the cases there was a neuritis or degeneration of the eighth nerve of unknown origin. The basis was probably anoxia, iscnemia, and altered metabolism. Emotional upsets preceding the onset of sudden deafness are emphasized in conjunction with the probable role of sludging of the blood. Blood sludging was found in all five patients examined for this condition and demonstrating sudden deafness. The author advises studying conjunctival vessels for blood sludge to determine the vascular role in sudden deafness.

Lindsay and Zuidema [32] found 16 cases with unilateral deafness of sudden onset. Four had definite causes, such as labyrinthitis and mumps. The other 12 were apparently healthy young adults with no obvious etiology for the hearing loss. The authors do not feel that vascular disturbances are the likely causes but that virus labyrinthitis is more probably the cause.

Doerfler [9] discusses psychogenic deafness, stating an incidence of 10 to 20% in military hearing centers. Certain hearing centers in civilian life are also encountering cases of psychogenic deafness. The discusser questions the value of audiometric retests as a method of detecting psychogenic deafness. He lists the following clues to the presence of a psychogenic loss:

1. Sudden unexplainable bilateral severe deafness perceptive in nature
2. A saucer-shaped audiogram (which was found in 80% of cases with psychogenic deafness in the author's experience)
3. Inconsistent audiometric and speech hearing results
4. Therapeutic response out of proportion to expectations
5. Results of special tests, such as the Doerfler-Stewart and the psychogalvanic skin resistance test

A classification for functional deafness is suggested by Harbert and Sataloff [33] under the following headings:

1. Psychologic
2. Malingering: (*a*) transient and (*b*) personality disorder
3. Psychoneurotic, including anxiety neurosis, conversion hysteria, and hypochondria

The authors suggest the following findings suggesting the presence of a functional hearing loss:

1. A history of onset of deafness which is contrary to clinical experience
2. No improvement in speech reception with amplification
3. Too great an improvement with minimal amplification
4. Marked fluctuation in a single test
5. Inconsistency in the results of several tests
6. Psychiatric manifestations
7. The presence of auditory reflexes

The authors indicate the usefulness of the psychogalvanic skin resistance test in diagnosing functional hearing losses.

Subcortical sensory aphasia, or pure-word deafness, with possible causes is discussed by Jones and Dinold.[34] A patient is presented with this condition resulting from a cerebral accident. Studies revealed word deafness despite satisfactory hearing and undisturbed language expression.

Reinhold [35] describes a case with normal hearing but with defective understanding of sounds, including speech and music.

Goodhill [36] presents a well-oriented and lucid evaluation of tinnitus. He emphasizes the psychosomatic factors and encourages their appreciation in treating a patient complaining of tinnitus. The scant definitive data available on tinnitus and the need for more research are made apparent in this excellent contribution. He [37] further makes an impressive attack on the heretofore impenetrable problem of tinnitus by adding to our thinking of this condition and demonstrating the ability to mimic tinnitus by producing certain sounds on records. Goodhill emphasizes the logarithmic progression of the decibel scale to explain that even a 5 to 20 db. above threshold tinnitus level can indeed be very distressing.

In a previous study Winston reported two subjects in whom hearing loss was discovered during the experimental administration of dimenhydrinate (Dramamine). In order to determine the reliability of this, Winston and Sataloff [38] rechecked the hearing in 46 subjects receiving dimenhydrinate. No effect on hearing was found. The importance of accurate audiometry and cautious interpretation of results is emphasized by this and other articles.

Saxon [39] believes that the most important pathology in presbycusis is not arteriosclerosis in the central nervous system but atrophy of the spiral ganglion, possibly accompanied by angiosclerotic degeneration of the inner ear.

The possibility of acoustic trauma resulting in children from cap-pistol and firecracker explosions is demonstrated in three cases.[40] These hearing losses are similar to those resulting from gunfire and are nonprogressive.

Saltzman [41] reports that 6 out of 12 patients on whom lesions were surgically produced in the mesencephalon or thalamus showed no significant audiometric change. In the other six patients a low-tone loss was the most constant finding.

One patient showed a V-shaped wedge in his audiogram. The author found a contraction of the auditory field in two of the cases. Recovery in auditory function of variable degree ensued in all of the cases with lapse of time.

Ménière's disease, according to Day,[42] is primarily a cochlear disturbance, and no vestibular symptoms occur until endolymphatic distension extends from the cochlea to the saccule and utricle. This extension may occur initially, or it may be delayed for years. Of 220 cases diagnosed Ménière's disease, 27 showed bilateral involvement, and of these, 23 had simultaneous involvement of the two labyrinths. It can be assumed that late or secondary involvement of the unaffected ear is rare. Of these 220 cases, deafness preceded vertigo in 83 and was not accompanied by any vertigo in 45 cases. In another 83 cases deafness and vertigo were noted together in onset. The main cochlear symptoms are fluctuation in hearing loss, a uniform loss of hearing throughout the scale, with decreased bone conduction, diplacusis, severe recruitment, and a characteristic low-pitched roaring tinnitus of a sea-shell type.

Fowler and Zeckel [43] suggest that Ménière's disease is primarily a psychosomatic disorder and that in advanced cases psychotherapy is required as well as medical help. They explain the condition on the basis of blood sludging associated with psychic phenomena usually present in patients suffering from Ménière's disease. In early cases, psychotherapy may reverse the course of the disease, and group psychotherapy should be considered, due to the similarity of significant traits of all affected patients.

One of the outstanding reviews in audiology is presented by Harris [44] in his brochure on loudness recruitment. He brings together in a coherent and teaching manner practically all that is now known of recruitment. He also summarizes 13 methods of determining recruitment and its clinical significance. The brochure should be read by audiologists working in this field.

From studying 32 cases of unilateral end-organ deafness and 16 of unilateral nerve deafness (most of which were established at operation), Eby and Williams [45] confirm earlier reports that little or no recruitment is found in the nerve-deafened ears and diplacusis is also absent. These, however, are present in ears with end-organ deafness. They also note that recruitment of speech intelligibility is present in nerve-fiber deafness but absent in end-organ deafness. In the latter condition there is a narrowing of the dynamic range for speech and a lowering of the threshold of discomfort. In normal ears the difference limen decrease with increased levels of sensation levels. In end-organ deafness the intensity difference limen remains the same, while in nerve-fiber deafness it decreases, as in normal ears.

Harris [46] stresses the need for investigating not only threshold levels but also the ability to perceive and judge the loudness of sounds whose intensities are well above threshold. In normal ears the loudness of a sound increases at a different rate from the increase in physical energy. This growth of loudness has been well specified for pure tones and tentatively for speech. In abnormal-hearing ears, however, the growth of loudness may take a different course from the course in normal ears, particularly in perceptive hearing losses. The term recruitment is applied to this phenomenon and is useful as a diagnostic tool. Using binaural loudness matching methods on patients with unilateral hearing losses, the author determined that matches of loudness for speech can be as precise as those for steady pure tones. Furthermore, irrespective of the type of deafness, recruitment of speech

follows a course parallel to that for pure tones. Four types of recruitment curves are distinguished, and the author gives three possible explanations for the delay feature of some of these recruitment curves. One explanation is that the so-called normal ear may itself have some recruitment.

Denes and Naunton [47] clarify the use of recruitment studies as a measure of cochlear function. The binaural loudness balance test is reliable and simple but requires one ear to be normal for the frequency being tested. Monaural loudness balance testing is cumbersome and difficult to evaluate. On the basis of difference limen testing, the authors devise a new method called the "D. L." test. They determine the intensity difference for loudness by applying two sounds of the same frequency to the same ear and by measuring the intensity difference between these sounds, which is just perceptible as a change in loudness. The difference limen is measured at two intensity levels; if the difference limen remains the same or increases with the intensity, then recruitment is present; if it decreases, then there is no recruitment. The results of this method agree with those of loudness balance methods and require between 5 and 10 minutes to do. A description of the necessary testing equipment is also presented.

Reger and Kos [48] expound on the measurement and implication of recruitment. They feel that it is highly desirable to make recruitment tests on all patients with non-middle-ear deafness. For uniform terminology they suggest the terms neural deafness and scalar deafness to designate pathology in the respective areas. Several tests are now in use to measure recruitment. The alternate loudness balancing test is limited to unilateral impairments. The equal loudness level contour is limited to persons with greater impairments over some part of the audiogram than another part. Neither test is useful in persons with bilateral flat equal hearing losses. Differential masking of pure tones and speech intelligibility curves are also used to measure recruitment. The test having the most wide application is the intensity difference limen test, since it can be employed with any type of unilateral or bilateral loss. Békésy initially described a new type of self-recording audiometer which records this intensity difference limen or the intensity range over which the test tones must be increased and decreased to be perceived and not perceived. Reger and Kos emphasize the importance of appropriate masking in testing unilateral losses. The authors have devised a complicated modification of Békésy's instrument with considerably more versatility. Their studies reveal certain temporary threshold shifts (TTS), which suggest the need for a reevaluation of the whole field of recruitment measurement under carefully controlled conditions. The discussion of this paper will also prove interesting to the reader.

Kobrak [49] describes three types of ear prosthesis: (1) ear-drum type, (2) tympanal type, which is placed in the niche of the round or oval window, and (3) ossicular type. The important prerequisites of the tympanal prosthesis are moisture and a fiber skeleton, like cotton, which maintains the shape of the moisture. The author makes direct observation of the tympanal prosthesis with stroboscopic light. An oxycholesterol-petrolatum ointment base (Aquaphor) is found to be the least irritating material to place into the tympanic cavity. When this covers the round-window niche, it aids hearing and can be left in the ear indefinitely. The ossicular prosthesis of Pohlman is described as excellent but mechanically difficult to maintain.

Kobrak,[50] using excellent photographic technique, also made movies of the physiology and pathology of the conductive mechanism.

O'Connor [8] describes the serious problems resulting from deafness in children. There are 276 schools or classes in which almost 21,000 deaf children between the ages of 3 and 21 are enrolled. Seventy-three are resident schools enrolling 13,636 pupils. He emphasizes the importance of the first five years of a child's life and the need for much earlier training than is now common. He notes that radical changes are occurring in many schools as far as the development of communication skills is concerned. Less emphasis is being placed on the traditionally formal approach to the mechanics of speech, and more is being placed on hearing aids and developing an early interest in communication values. He states that there are very few so-called completely deaf children and urges the use of residual hearing whenever possible. Most of the pupils in schools for the deaf have losses in excess of 90 db. He answers some very pertinent questions, such as "Can the deaf really learn to speak?" The answer is yes, with a suggestion that otologists visit their nearest school. The efforts spent on oral education are definitely justified. He makes a clear-cut distinction between the deaf and the hard of hearing and feels that the latter group should be educated in or close to regular school programs for the normal hearing. A child with less than 60 db. loss can satisfactorily get along in a normal program if he receives such things as favorable seating, lipreading, a hearing aid, and individual assistance.

Goodhill,[51] in an excellent study, introduces the significance of Rh incompatibility as an etiologic factor in infantile nerve deafness. He [52] further presents a careful study to show that bilateral nerve deafness may be produced by erythroblastosis in athetoid cerebral palsied patients. He suggests that the lesion is in the nuclear masses as a result of kernicterus.

Kinney [53] reviewed all the published volumes of *The Quarterly Cumulative Index Medicus* and found 42 articles concerning the pathology of hereditary deafness. Confusion between deafness and hardness of hearing and the paucity of pathological material are the two most outstanding impressions. Kinney suggests a classification of congenital deafness divided into five categories:

1. Hereditary
2. Malformations
3. Maternal occurrence during first trimester of pregnancy
4. Intrauterine accidents
5. Birth injuries

Two kindred showing nerve deafness are reported by Dolowitz and Stephens.[54] In kindred I a definite pattern of inheritance was indicated as an autosomal dominant with complete penetrance. In kindred II nerve deafness occurred only in the males and was due to a sex-linked recessive gene or is a secondary effect of pyelonephritis. It is clear that there is more than one mechanism producing "hereditary" progressive nerve deafness of later life.

Goodhill [51] studied 15 erythroblastotic infants, 77 cases of infantile perceptive deafness, and analyzed 904 questionnaires concerning profoundly deafened children. He suggests the following classification of infantile perceptive deafness: (1) hereditary and (2) acquired. Under the latter he places the following types:

1. Prenatal (infections, drug intoxication, and other acquired developmental anomalies)
2. Natal (birth injuries and erythroblastosis fetalis)
3. Postnatal (meningitis, viralacoustic neuritis, encephalitis, and miscellaneous causes)

The significance of Rh incompatibility as an etiological factor in producing infantile deafness is elaborated. Of the 904 questionnaires, about 27 probably demonstrated Rh incompatibility as an etiological factor. The author predicates the deafness as being nuclear and resulting from the toxic effects of kernicterus. He states that although the Rh factor does not play a major role in infantile perceptive deafness, it is, nevertheless, a significant one and may point to other serologic factors as etiologies. He also reviews the literature on maternal rubella as a cause for infantile deafness and describes 19 personal cases as well as 186 cases obtained from the 904 (about 20%) questionnaires. The author stresses the public health role in recognizing this condition for preventative measures.

The importance of early diagnosis of hearing loss is emphasized by many authors in order to aid speech development. Hearing aids should be recommended at the age of 2 years or younger.[55] Hard of hearing children should be educated with normal-hearing children and helped by the aid of speechreading and auditory training.

Hardy and Bordley[56] give a detailed description of the psychogalvanic skin resistance test which they developed at The Johns Hopkins University. The technique is exacting and requires a team to perform. It is not adaptable to the clinician's office and is only one part of a comprehensive hearing evaluation for infants and very young children.

Hardy and Pauls[57] state that a two-man team is one basic requirement for a psychogalvanic skin resistance test in small children in order to get good results. There is also a great need to control the environment of the test situation to reduce the array of stimuli and stabilize the child's behavior to obtain the best results. A play room is essential to first put the child at ease before the test. A gross evaluation of the child's hearing should also be done before the psychogalvanic skin resistance test by means of a soundproof room and speech-reception equipment. It is essential to have equipment out of sight of the child during the test. It is advisable to permit only one parent (out of sight of the child) in the testing room. Children of different ages should be given different-sized chairs in order to put them at ease. Electrodes are placed either on the finger tips, palm and back of hand, or on the foot, if necessary. The shocker is always placed on the calf of the leg, and all electrodes should be firmly attached. Pretest studies of dermometric measurement will make the test easier to read. It is extremely important to handle the child properly during the test so that a steady graph is maintained, except for the introduction of shock or sound stimuli. The conditioning process is the key to the test situation. A light faradic shock is given three or four seconds after a sound stimulus in order to condition the patient. The need for three or four seconds delay is to give the shock stimulus one-half to one and one-half seconds to record a change in the sweat glands. An initial tone of 500 cps at a level well above threshold is to be used. This threshold is based on the results of the gross testing done before. Short bursts of test tones are more stimulating, louder psychologically, and less fatiguing than long-sustained ones. The process of tone and shock is repeated until the patient is conditioned, and then just tones are used, gradually diminishing toward threshold. Occasional shocks can be used in order to refresh conditioning, as determined by the tester.

Neither the shock nor the test itself is traumatic when the test is handled properly. The success of the psychogalvanic skin resistance test is not accomplished

by reading about procedures but, like the success of the surgeon, requires much experience, and for this reason it is doubtful that this test will ever become routine procedure by physicians or in a school system. Masking may be used when two ears differ by 20 to 25 db. The psychogalvanic skin resistance testing cannot be done on children under dosage with any of the barbiturates, and patients must be free from drugs for 48 hours prior to testing. Children with overactive sympathetic nervous systems may be given ¼ grain (15 mg.) of phenobarbital (Luminal) 20 minutes before testing.

Hardy and Bordley [58] report on the high degree of reliability found after retesting some of the 650 subjects tested with the psychogalvanic skin resistance test. This outstanding contribution to audiology utilizes the Fere effect and a modified form of Pavlovian conditioning. The child is conditioned to sound, a mild faradic shock from an inductorium being used. The conditioning is accomplished in about 8 to 12 administrations of a short burst of test tone followed in about three seconds by a shock. The validity and reliability of this method, when done properly, are now established.

Dix and Hallpike [59] review the operation of the peep-show equipment. Certain psychological and technical difficulties have presented themselves during the testing of some 350 subjects with this method. The preparation of the child prior to testing is extremely important. The child should not be hungry, tired, or apprehensive; the atmosphere should be one of play. It is unwise for the testers to wear white coats or to try to give an otological examination at this time. Confidence must be established. Certain visual, motor, and mental defects make this technique unsuitable. During the operation of the tests the intensity of the initial stimulus is important. To be able to provide the correct intensity requires experience, but usually an intensity of about 80 db. is suitable. The duration of the test procedure is usually 12 to 15 minutes. The results of this testing usually are adequate for making recommendations for special educational measures. In a follow-up of some 40 of those children who had received special education as a result of the testing, the validity of the test was confirmed. The authors also emphasize that for the majority of deaf children pure-tone audiometry provides a useful measure of hearing capacity for speech.

The necessity for early and accurate diagnosis of hearing loss is also stressed by Guilford and Haug.[60] The shortcomings of the following available techniques are described:

1. Ewing electric train tunnel test situation is cumbersome and lengthy.
2. Bloomer picture-card test is lacking in attention appeal and may be confusing.
3. Dix-Hallpike peep-show test lacks dramatic attention-holding appeal and does not test unilateral impairments. It also requires elaborate equipment.
4. Marcus-Gibbs electroencephalography technique is costly, is complex, and requires special technicians and surroundings.
5. The psychogalvanic skin resistance test is extremely costly and requires special test surroundings and technicians. Criticisms are also made that below 50 db. poor responses are obtained and that inconsistency of response is frequent.
6. Noise-maker tests are qualitative and not quantitative and are given in free field.

The authors introduce their own apparatus, the Pediacoumeter, and describe its use; it was designed in an attempt to overcome the shortcomings of most of the other tests. This instrument is an electronic portable attachment to any conven-

tional audiometer, with seven jack-in-the-box heads representing the pure tones tested. A very appealing child's story is used in youngsters who can understand directions. They are told to let the little people out of their boxes when they hear a cry for help to get out. Electronic controls prevent indiscriminate responses from the patient and the jacks-in-the-boxes. In children who lack understanding of directions, conditioning is used. Test and retest situations were remarkably consistent.

Marcus [61] feels that in examining a child suspected of having a hearing loss the maxim should be followed that the child is hard of hearing until proved otherwise. He describes the use of an electroencephalogram as a qualitative method for determining the presence of hearing in young children and infants. The child must be asleep and be subject to an arousal response as a result of auditory stimuli. The change in the electroencephalogram pattern, resulting from the auditory stimulation, is indicative of residual hearing. During the testing of 53 children in whom clinical evidence of severe hearing loss was found, 19 showed electroencephalogram evidence of an intracranial disorder. This also was found in 9 out of 18 children with slight hearing loss, who presented delayed, retarded, or unintelligible speech. The author concludes that disturbed cerebral control appears to be a factor in delayed, absent, or unintelligible speech.

Referring to Marcus' difficulty in doing quantitative audiometry with an electroencephalograph because of the complications produced by introducing another electrical circuit on the delicate one of the instruments, Gidoll [62] reports a successful audiogram on an 18-month-old child. He also uses an arousal response during light barbiturate administration (inducing sleep), and interference on the electroencephalograph circuit is avoided by grounding the standard audiometer used as a calibrated sound stimulus.

Testing small groups of children using magnetic receivers connected to both air and bone jacks of an audiometer makes possible quick screen testing since the bone jack is automatically 50 db. more than the air jack.[63] Raising of hands instead of paper scoring is just as accurate if the operator employs the cutting-out of several receivers at will to check the validity of each child's response.

Nielsen [64] shows that group testing of school children by pure-tone audiometry, using earphones, long and short signals, and charts for each child to record what he hears, makes it possible to test a community of several thousand every other year. Hearing losses in the youngest school children are detected early enough to minimize severity in hearing losses in upper grades.

Wishart [65] criticizes the separation of most audiology clinics from the ear, nose, and throat clinics and feels that extravagant centers are needed only for research and not clinical use. Both the hearing testing and the treatment should be done by the otologist. A pleasant relationship between the patient and otologist is essential. For testing little children, the author finds the peep show an invaluable first approach. After this a Melody Master is used for teaching the child to be tested on the speech audiometer. The value of the psychogalvanic skin resistance test is being investigated in the author's hearing clinic. He finds no use for watch tests, whisper tests, tuning-fork tests, or the monochord. The author advises against the use of free-field testing in any hospital clinic for the hard of hearing, primarily because live voice is used, and this is hardly possible to standardize. He feels that live-voice testing for "measurement" is a delusion. He also indicates

that one cannot infer a patient's hearing of speech from a pure-tone audiogram. In his own testing of children Wishart makes all tests short, believing that a young child ought not to be in the soundproof room longer than a few minutes at a time. Wishart criticizes the assumption that every lesion of the perceptive apparatus is incurable and describes a 6-month-old patient with a diagnosis of deafness, who started to put three and four words together at about 2 years of age.

The importance of utilizing certain psychological testing programs by audiologists is suggested by Myklebust.[66] These would cover the mental competence, social maturity, personality, motor ability, and educational achievement of the patient.

According to Hedgecock,[67] an increasing number of agencies are providing improved facilities for dealing with the hearing problems of children. These include colleges, medical schools, societies, and private groups. The author mentions various tests for determining hearing loss in children and emphasizes the importance of the psychogalvanic skin resistance test in experienced hands. An accurate and quantitative evaluation of hearing is an important first step in bringing the parents to grips with the problem. It is the only available hope for ending the frequently futile search for a diagnosis of normalcy and preoccupation with unrealistic hopes for curative treatment. A forthright approach is the only one that is just to the parents and child. Appreciation of the problems of both parents and child and an optimistic, calm assurance by the otologist are essential for a satisfactory family relationship. The basic emotional needs of the handicapped child are the same as those of all children. The two most vital are (1) to be loved and to love in return and (2) to strike out against situations which are unpleasant. These can be provided by understanding parents along with adequate training. In deciding what kind of a school to send the child, the parents should keep the welfare of the child in mind more strongly than their own desires. This is a profound article and well worth reading.

Mrs. Spencer Tracy [68] emphasizes the importance of communication in daily life and the handicaps resulting from deafness. The deaf child can learn to talk intelligibly and communicate well with proper training. This training should be done as early as possible and, ideally, through the parent and in the home. The work of the Tracy Clinic is to find, encourage, and help guide and train parents of deaf and hard of hearing children. The clinic not only gives information, material, and methods to parents but is just as concerned with feelings too. The clinic holds classes for parents and also has a nursery school in which the whole family participates. The most important service is the famous correspondence school. Mrs. Tracy makes a sincerely profound appeal for the early detection of hearing loss and a positive, constructive program for these children.

Jellinek [69] also stresses the importance of early diagnosis and training for the hard of hearing child. If there is any doubt of the child's hearing ability, auditory training should be instituted to better the chance for speech development. A hearing aid is desirable, even at 18 months of age, so that the child can hear his own voice and stimulate speech development through comparison with others. The author believes that 40 to 50% of deaf children could attend ordinary public schools if adequate early training is instituted. Children with mild hearing losses should be treated as normal children. Most children enjoy and are proud of their hearing aids but often have to be gradually accustomed to their use.

Landis [70] states that in every classroom there are from one to three children who have defective hearing. This is about 3 to 4% of the country's school children. His survey in Pennsylvania revealed almost 50,000. School children were tested in the 1st, 3rd, 5th, 7th, 9th, and 11th grades. Those with a 20 db. loss in two or more tones in an ear were referred to a local otologist through their parents. There are over 1,000 deaf children in schools for the deaf in Pennsylvania, and the cost of each case amounts to $1,800.00 a year. Landis encourages the use of residual hearing and the education of hard of hearing children in schools for normal children. He urges early training for the preschool child.

A study [71] of 20 children using hearing aids between the 4th and 10th grades reveals that (1) the gain in speech reception with the aid had little relation to the amount of daily use; (2) at these ages girls wear their aids more significantly than boys; (3) the amount of residual hearing seems to have no relation to length of daily use of the hearing aid.

In a study [72] of the relation of cleft palates and hearing, middle-ear pathology was found in almost all of 30 children with cleft palates and measurable hearing loss in the great majority. Further studies are indicated.

Hirsh [73] presents a correlation between the clinical and physical aspects of pathology in speech communication. He emphasizes the importance of "auditory feedback" for the control of speech production and explains how hearing impairment may affect the intelligibility of the subject with such a handicap. Deaf children who cannot hear their speech by auditory feedback use their kinesthetic system as a substitute.

From a series of 17 patients who sustained blows to the head, Schuknecht [74] indicates that deafness may result irrespective of any evidence of fracture in the temporal bone. The loss is generally of the nerve type and most prominent at 4000 cycles. This loss very frequently occurs in the contralateral ear to the injury. Audiograms and histories are presented.

Schuknecht and his associates [75] correlated deafness and inner-ear pathology by subjecting 10 conditioned cats to head blows and studying hearing and anatomical specimens. Most of the losses in hearing were around 4000 cycles, and middle-ear or brain damage was ruled out by microscopic examinations. The primary site of damage was in the lower portion of the organ of Corti. The degenerative changes in order of severity were (1) loss of external hair cells; (2) loss of external and internal hair cells; (3) flattening of the organ of Corti; (4) complete disappearance. There was also secondary degeneration of nerve fibers and ganglion cells supplying the degenerated areas in the cochlea. The authors conclude that a pressure wave from the trauma is transmitted to the organ of Corti, causing a violent displacement of the basilar membrane and injury.

Perlman [76] demonstrated in 30 guinea pigs that experimental occlusion of the inferior cochlear vein did not produce cochlear or vestibular dysfunction, as determined by cochlear microphonics, action potentials, nystagmus, and head and trunk torsion.

In presenting "Recent Investigations of Sound Conduction" on the normal ear, Lawrence [77] very lucidly explains the mechanics of sound conduction and the role of the middle ear from three aspects: (1) the matching of properties of air to perilymph, (2) the transmission of periodic vibrations with minimum alteration,

and (3) protective mechanism. Lawrence points out that the ear drum and ossicles in the normal ear prevent a loss of about 28 db., which would occur if impedance matching of air and cochlear fluids did not occur. He also shows that the natural frequency of the middle ear is near the middle of the audible range, providing maximum sensitivity in this region. High damping also helps make the response uniform with frequency.

Wever [78] correlates hearing loss and pathology in the conductive mechanism. The hearing loss resulting from a perforation in the drum membrane varies, of course, but several related functions are presented. The perforation directly reduces the area of the membrane which responds to the pressure of the sound wave. The perforation also allows the sound pressure to act on the inner surface of the drum and oppose the pressure on the outer surface. Since the manubrium is the direct attachment to the drum membrane and transmits the motions, a perforation in this area is particularly serious. Finally, a perforation reduces the stiffness of the middle-ear mechanism and consequently produces greater loss in hearing for tones above about 1000 cycles. The author demonstrates that interruption of the incudostapedial joint (in an otherwise normal cat's ear) produces a loss of about 60 db. If, however, the drum membrane and outer two ossicles are also removed, the loss is not so great by about 15 to 20 db. Fixation of the ossicular chain may occur in at least three forms: by (*a*) fluid, (*b*) mechanical fixation by scar tissue, and (*c*) otosclerosis. The characteristics of each type are well described and explained. The major portion of this excellent article concerns itself with otosclerosis.

Reudi [79] feels that maintenance of normal range of oxygen pressure is just as important as the difference in air pressure between the middle and outer ear in aerotitis media. He also states that ear defenders utilizing selective sound filtration may be completely effective in preventing acoustic trauma. His studies include tests with animals as well as military personnel.

Using cochlear potentials in cats, Wever and Lawrence [80] found that the middle-ear apparatus presents uniform properties to sounds over a wide frequency range. With mechanical and electrical models, they determined that this apparatus is able to act uniformly as a mechanical transformer due to two factors: the development of two mechanical resonances about two octaves apart and the presence of a high degree of frictional damping. These help explain why the middle-ear system causes so little disturbance in the sound patterns which it receives.

In a further investigation of the transmission properties of the stapes in the cat, the authors, Wever and Lawrence,[81] conclude that in the lower frequencies the stapes improves the mechanical tuning of the ear but only adds complexities in the uppermost frequencies.

Wever [81] repeats an earlier finding [78] that there is little difference in effectiveness in sound going into the round or the oval window, with only a slight difference in favor of the latter, due to the stapes. In the normal ear, this phenomenon, which may result in cancellation of waves in the cochlea, is unimportant because the ossicular chain selectively and almost exclusively conducts the sound to the oval window.

Loss of hearing, predominantly in the high tones, may result from accumulation of fluid in the middle ear. This occurs in the early phases of fluid accumulation, due to increased mass and damping. Later, stiffness is also increased, and all tones are greatly affected.

Wever explains the paradox of many patients with observed pathological changes in the ears and normal hearing. The subjective tests for determining auditory threshold are often not fine enough to detect changes in loudness more closely than 5 or 10 db. Since these are logarithmic terms, the change may be as much as 40% without being determined on the routine audiogram. He feels that all pathological changes in the middle ear probably affect hearing to some degree. He further states that, from fixation of the stapes by otosclerosis extending over a number of years, hearing impairment may exceed 80 db. and that fenestration may produce a dramatic return of hearing despite these profound degrees of impairment.

New methods of blocking the round window with wax have practically the same results as those obtained by blocking the oval window. The analogy to otosclerosis is implied. The author explains that in the fenestrated ear sounds enter the cochlea simultaneously by the round and oval windows but not equally and not in the same phase; otherwise the sounds would cancel themselves. Perforation in the tympanic flap impairs the phase, and partial cancellation occurs, producing impaired hearing until the perforation heals.

The role of the tympanic membrane for the reception of sound has led to considerable controversy. Payne and Githler [82] evaluate the effect of size and location of perforation, using cats and studying cochlear potential responses. They conclude that no matter where the perforation is located a loss occurs for every frequency. Small lesions in the posterior region cause the greatest response loss in the frequencies below 2000 cps. Anterior perforations cause the next greatest loss, and perforations around the umbo cause the least loss for these lower frequencies. Increasing the size of the perforation does not alter the general shape of the response function until almost the entire tympanic membrane is gone.

Munson and Wiener,[83] using very fine methods to measure sound pressures in external ear canals, conclude that often the variability in subjective responses may be due to inadequate technique for measuring the levels of the stimuli. They imply that much more constant and reliable responses could be obtained from hearing tests if the stimulus or source of sound could be more accurately controlled and measured. The importance of using calibrated equipment and controlled conditions in clinical and experimental audiology is certainly a significant factor.

During 1950, four different hydrodynamic theories were introduced to explain the action of the cochlea. Zwislocki,[84] Ranke,[85] Peterson and Bogart,[86] and Fletcher [87] presented very complex theoretical explanations of cochlear function. All of them proved the usefulness of their equations by using the experimental data obtained by G. von Békésy. Most of the authors demonstrated close agreement between experimental data on localization and phase velocities and their theoretical calculations.

Wever and Lawrence [88] attempt to crystallize the rather complicated and numerous theories to explain the conduction of sound in the cochlea. The two principal types are the fluid and the membrane theories. The former proposes that the energy from the sound pulse finds its way to some particular region of the basilar membrane directly by way of the cochlear fluid. The latter type of theory proposes that the conduction is principally by a wave motion in the basilar membrane or, sometimes, in Reissner's membrane. A useful analogy to this latter theory is the traveling wave set up by a rope which is attached at one end and given a quick flip on the other end. Recent interest in the traveling-wave theory has been so

enthusiastic that there are now 12 different theoretical explanations offered. The experimental findings of Békésy in 1928 are generally the basis for all these theories. The basic principle of the traveling-wave theories is that all regions of the membranes beyond the basal end receive their vibratory energy from regions of the membrane lying just below them along the cochlear spiral. The cochlear fluid plays only a secondary role in this process. The authors list the features which practically all the theories have in common and then present their own experimental findings on cats. Their results are out of harmony with the traveling-wave theory and in agreement with a local action theory which is also consistent with Békésy's results. The authors suggest that their volley theory can explain facts of pitch perception and other auditory experiences which other theories have failed to do.

Wever [89] uses loudness and pitch discrimination data from various sources to further his claim that the perception of low-frequency tones is dependent on the frequency—not the place—principle. The distance between the maximum of deformation for a 100-db. 1000-cycle tone 52% from the basal end of the basilar membrane and the maximum point for an 860-cycle tone 48% away represents 1 jnd (just noticeable difference) in loudness but 55 jnd's of pitch. Likewise, the distance between the maximum for the 1000-cycle tone and the maximum for a 1200-cycle tone 56.4% from the basal end represents 1 jnd of loudness but 70 jnd's of pitch. According to assumptions, the intervals 1000 to 860 and 1000 to 1200 should be just discriminable for pitch. Similar calculations for higher frequencies turn out better, but for lower frequencies they turn out even worse.

Wever assumes that the pitch and loudness discriminations for the intervals mentioned depend on the "same nerve fibers" and thereafter uses the frequency principle for loudness to discredit the place principle for pitch. As Wever has explained elsewhere,[90] pitch for the place theory "depends upon the vibration of the particular portions of the basilar membrane, and hence the excitation of the particular nerve fibers (within each respective portion), whereas loudness for the frequency theory depends on the number of nerve fibers set into operation by the "areal spread of action along the basilar membrane."

In regard to the relative roles of the two principles in the evolutionary development of the ear, Wever points out that primitive ears operate as frequency receptors and that the place principle came to supplement the frequency principle as a consequence of anatomical and physiological changes in the cochlea during its later stages of development.

Huggins and Licklider [91] analyze the various theories that attempt to reconcile the "broadly tuned" maxima of vibration of the cochlear partition with the place principle. Experimental data show that the neural channels are narrower than the cochlear channels, thereby implying that the excitation of the auditory nerve is governed by mechanical and/or neural sharpening mechanisms. In all the theories, the maximum displacements are caused by traveling waves, but the mode of excitation is believed to be due either to eddies in the perilymph (Békésy's eddy hypothesis), the interaction of the displacement amplitude and the phase velocity of the wave (Ranke's velocity-tuning hypothesis), the maximum slope of the wave found by differentiating the maximum amplitude as a function of its distance from the stapes (gradient hypothesis), distortion of the bodies of the hair cells by the

curvature of the cochlear partition (curvature hypothesis), or to the tension exerted on the hair-cell cilia imbedded in the beam-like tectorial membrane (Huggins' beam hypothesis).

Békésy's observations concerning the strengths and dimensions of the eddies and their behavior as a result of complex stimulation were interrupted by the war. The case for the gradient hypothesis is weakened by the relatively low magnitude of the slopes, the small displacement of the hair cells, and the lack of concentration of the shearing forces on the partition. Small hair-cell deformation speaks ill for the curvature hypothesis.

The double differentiation hypothesis introduces the concept of neural sharpening from the positive and negative second derivatives of the vibration envelope—positive being associated with excitatory neurons and negative with inhibitory neurons within any segment of the cochlear partition. With the weighting function introduced as an analog of the neural process, the evidence seems to support approximate differentiation as a factor in sharpening cochlear frequency analysis.

It may be that a number of tranforms of peripheral stimulation are presented simultaneously to the higher centers, and the cortex either chooses the most useful or samples them all to reconstruct the original message.

From Békésy's experimental data, Fletcher [92] calculates for different frequencies the displacement amplitudes and phases of the basilar membrane, the velocity of propagation of the traveling wave at each point on the membrane, and the volume of fluid displaced at the stapes per dyne pressure. Dividing the basilar membrane into small square vibrating elements (the side of each unit being equal to the width of the membrane at any point), he calculates their mass, stiffness, resistance, average velocity, and the pressure acting upon and the force driving them.

The force driving any one element is equal to the product of the pressure exerted at that point by the vestibular and tympanic canals times the square of the width of the basilar membrane, and the average velocity of the element is equal to this product divided by two times the mechanical impedance. From his hydrodynamical equations, Fletcher assumes that the velocity of sound in the fluid is very much greater than the velocity of propagation of the wave traveling along the basilar membrane. Thus the fluid may be considered incompressible, and its displacement by the stapes is registered as an equal displacement at both the round window and points on the basilar membrane—except for frequencies below 200 cycles, where some of the fluid passes through the helicotrema.

For some frequencies, the phase angle between the stapes displacement and the basilar membrane displacement indicates that the membrane behaves like a resonance system. However, as Zwislocki [93] has pointed out, any resonance theory of hearing is to be discounted, since the point of maximum amplitude would have to be at the place of shortest wave length. Not only are the damping vibrations nearly aperiodic, but the cochlear partition shows no resonance for frequencies below 1000 cps.

Because of low mechanical impedance due to lack of rigidity of the basilar membrane toward the apical end, the middle ear acts as a poor mechanical transformer at low frequencies. The amplification ratios computed from Békésy's experimental data show that amplification occurs for frequencies above 650 cps but that below this frequency there are very large attenuations—as much as 30 db. at 100 cycles.

Fletcher [94] uses the equations developed in the previous paper to prove that the poor transformer action of the middle ear is responsible for the variation of acuity of hearing with frequency. He assumes that the discharge rate of a unit length of the basilar membrane near the hearing threshold is proportional to the energy of vibration or the velocity squared, the density of nerve endings, and the impressed frequency for frequencies up to 300 cps—for which the nerve is excited by the maximum or minimum velocity during each cycle. Above 300 cps, the rate is dependent on a constant and is independent of the frequency because of the refractory period of the nerve. Putting these assumptions into an equation, he computes the threshold values of loudness for frequencies in the range 25 to 6000 cps and finds that they could adequately account for the variation of acuity with frequency.

In a classic investigation, Hilding [95] studied the origin and make-up of the tectorial membrane in fresh specimens and established its attachments and actions. He presents a new concept of the translation of broad sound vibrations in the cochlear fluids into a localized stimulus applied to the organ of Corti. The mechanism involved is a shearing force applied through the tectorial membrane and transmitted on only one spot to the organ of Corti. This spot is located where the shearing force is directed in the axis of the relatively firmly fixed and nonflexible fibrils. The author demonstrates the anatomical application of his concept with conviction. This paper should be read by everyone interested in the physiology of hearing.

Davis [96] points out that a good physiologic theory requires accurate anatomic knowledge as a foundation. The relation of the nerve fibers to the hair cells is fundamental to any theory of the excitation of the auditory nerve. Both internal and external hair cells have radial and spiral nerve fibers. The radial fibers form the chief nerve supply of the inner hair cells but are in minority to the external hair cells. The spiral fibers run from considerable distances in both directions along the organ of Corti and give off many branches. These spiral fibers form the "external spiral bundles." The innervation of the hair cells is complex. The spiral fibers to the internal hair cells seem to be efferent with cell bodies in the superior olivary complex of the opposite side. They run from this nucleus in the tract of Rasmussen and form a compact bundle in the auditory nerve. This bundle and the fibers of the tract of Rasmussen do not degenerate, like other auditory fibers, when the organ of Corti is destroyed by severe acoustic trauma. Davis also discusses excitation in sense organs, cochlear microphonics, and the summation potential. He pictures the cochlea as a delicate acoustic analyzer that localizes the energy of the sound in different areas according to the frequency of the sound waves. It is also a delicate electrochemical apparatus.

Many major contributions in audiology have come from the experimental neurologist. The principal aim of this scientist, as conceived by Galambos,[97] is to discover the physiologic correlates for psychological phenomena. He emphasizes that in audiology the search is primarily for neural correlation of sound frequency and intensity to explain pitch and loudness sensations. Some important psychological phenomena requiring explanation in the nervous system are the differences in stimulation with speech as opposed to pure tones, localization of sound, and pathological conditions of hearing. These and other problems are being attacked by different methods by experimental neurologists, physiologists, psychologists,

acoustic physicists, anatomists, otologists, and many others. Galambos expresses the hope that eventually a correlation between physical and subjective patterns can be established and thus provide a rational basis for predicting an observer's responses to simple stimuli. He clearly summarizes the role of the auditory nervous system and emphasizes that new anatomical connections are still being uncovered, such as the connection between the cochlear nucleus and the cerebellum, and the cochlear nucleus and the superior olive of the opposite side. Galambos and his associates have also demonstrated physiological evidence of a pathway between one cochlea and its mate on the opposite side.

Readers desiring a clear description of the microelectro technique should read Galambos' article.[97] From studies with this technique the author has demonstrated that neural elements in the auditory pathways at the cochlear nucleus and medial geniculate body respond to a rather specific frequency at threshold. If the intensity of the stimulus is raised, however, the same unit can be activated by a wider band of frequencies. The author has also shown in cats that at the cochlear nucleus certain tones may inhibit neural activity. This may possibly lead to a better understanding of how the human ear can so well distinguish between two tones close together in frequency.

Lawrence and Wever[99] show that extreme oxygen deprivation diminishes electrical cochlear potentials generated by sound stimulation. The deterioration of potentials due to anoxia seems to be independent of frequency. Histological examinations of the animals used show cochlear deterioration in all and a greater deterioration in those exposed to most extreme anoxia. The pattern of deterioration is irregular but more pronounced in the basilar end. The stria vascularis has no relation to the state of the organ of Corti in the immediate vicinity. The supporting cells succumb first, while the hair cells are well maintained into later stages of degeneration.

Wing and his associates[100] record cochlear microphonics from the round window of the ears of cats breathing different mixtures of oxygen in nitrogen. The generators of cochlear microphonics are thought to possess a very limited store of available energy for anaerobic function and are much more sensitive to hypoxia than has hitherto been believed.

Exploring the possibility that the cochlear microphonics may be due to a system in unstable equilibrium, i. e., physiological (chemical) phenomenon rather than physical, Békésy[101] tries to determine whether the electrical energy in the cochlea of a lightly anesthetized guinea pig exceeds the mechanical energy due to displacement of the cochlear partition. Placing one electrode near the stapes footplate (zero point) and an indifferent electrode at different points in the perilymph, Békésy reports D.C. potentials all along the vestibular and tympanic canals, indicating a continuous current flow in the cochlea. Then shifting the electrode to the round window, he finds that the positive potential relative to an indifferent electrode becomes smaller (D.C. fall) upon application of a sound stimulus of constant intensity, but with a simultaneous proportionate increase in the size of the A.C. potentials (microphonics). When the sound is turned off, the microphonics return to their original value, but it requires a long time for the original value of the D.C. voltage to be restored, indicating a consumption of electrical energy in the cochlea.

Repeated displacement of the cochlear partition by an iron ball (electromagnetically controlled) reveals that the electrical energy consumption of the microphonics is not accompanied by any "mechanical" energy losses, since the damping of the free oscillations of the partition remains unaffected. Moreover, when a needle point is pressed against the Reissner membrane (single static deformation), a continuous potential is produced (D.C. microphonic), which disappears with the removal of the needle. Békésy's calculations show that the electrical energy consumption produced by the pressure on the membrane is greater than the mechanical work done, indicating that the mechanical energy of the stimulus is not solely responsible for the electrical energy of the microphonics.

Using acoustic clicks as stimuli, Rosenblith and Rosenzweig[102] observed that by placing the active electrode at the bulla of the cat rather than at the round window areas are found in which the microphonics are attenuated and even canceled, thereby permitting observations of the neurals without interference. When the position of the electrode is shifted, preferential treatment is given to certain components of these action potentials. Potentials originating at lower levels of the auditory nervous system are easily recorded with the electrode on the surface of the brain (dura mater). Even on the intact skull cortical and subcortical responses are obtained (identical in latency and behavior) which register almost as well as those on the brain surface. Placing the electrode at one bulla while the contralateral ear is stimulated reveals that the responses for the two ears are opposite in polarity. On the skull, however, the polarity of the two responses tends to be the same.

When the same ear receives two clicks in rapid succession, the response to the second click shows a great reduction in amplitude (interaction). With the electrode at the left bulla, no evidence of binaural interaction seems to exist when the left ear is stimulated just 3.6 milliseconds after stimulation of the right ear. The authors express their intention of studying the problem of dichotic interaction and auditory localization on other animals—both awake and anesthetized—using as stimuli clicks and pure tones of all frequencies.

In the above experiment, Békésy noted that with increasing anoxia the size of the D.C. fall begins to decrease later than the neural potentials and the microphonics, indicating that the D.C. potential drop is a basically different process from the Wever-Bray potentials. He also noted that without acoustic stimuli the potential difference between the vestibular and tympanic canals increases with minute perforations of the Reissner membrane and inferred that certain structures within the cochlear partition must register greater "resting" potentials than are found between both canals.

Exploring this inference further, Békésy,[103] at a point in the scala vestibuli 15 mm. from the stapes footplate, inserted an indifferent microelectrode just above Reissner's membrane and pushed a second micropipette through the cochlear partition at various depths. To prevent damage to surrounding tissues, the micropipette cannot be larger than 0.3 μ in diameter and must be moved exactly parallel to its axis. Potentials read 20 mv. during penetration of Reissner's membrane, 50 mv. in the endolymphatic space, –40 mv. during penetration of Hensen's and Claudius' cells, and –2 mv. once the basilar membrane is perforated. Higher potentials are obtained with greater care in perforation. There is a very high potential

difference between the endolymph and Corti's organ, and near the round window the endolymph registers as much as 80 mv. with respect to the perilymph in the scala tympani.

These measurements include the additional few millivolts supplied by the junction potentials between the fluid in the two electrodes and the perilymph and endolymph—the exact sizes of which are difficult to calculate.

Békésy's [104] next experiment on guinea pigs determines the insulating properties of the various membranes of the cochlear partition, the electrical conductivity of the fluids in the cochlea, and the approximate place of origin of the first-order (most sensitive) microphonics.

With an electrode each in the vestibular and tympanic canals connected to an oscillator, a potential difference is introduced across the cochlear partition. A third indifferent electrode is inserted just above the basilar membrane, with an automatically controlled fourth perforating electrode close by. These two electrodes are connected to D.C. and A.C. recorders. Perforation of the cochlear membranes from the tympanic side produces the same results for the D.C. potentials, as described in the preceding paper. However, for A.C. voltages there are no negative potentials inside the different cells but continuous jumps in positive potential as the perforating electrode goes through the basilar membrane, the endolymphatic space, and Reissner's membrane. The over-all potential increase is the same across the basilar and Reissner membranes, indicating an equal insulation of the endolymph from the perilymph on both sides.

Except for the slight positive phase shift in the endolymphatic space, there is a continuous negative phase shift with respect to the oscillator's phase during the entire perforating operation Greater phase shifts occur with increases in frequency or when the indifferent electrode is placed in the body just outside the ear, indicating that the body acts like an electrical ground system connected to the endolymph through the brain and the auditory nerve. The several impedances can be measured through this ground. The endolymph acts like a pure resistance, whereas the membranes are capacitors with a shunt resistance. Essentially the same results are obtained when acoustic stimuli are used instead of the oscillator.

Changes in resistance of the perforating electrode with respect to the surrounding fluid seem to indicate no significant difference in conductivity between the endolymph and the perilymph. Because of the phase reversal and rise in size of the microphonics when the organ of Corti and the basilar membrane are disturbed by the penetrating electrode, it appears that the main source of the first-order microphonics lies on the basilar membrane rather than the Reissner membrane.

The cochlear partition may be said to be composed of three types of tissues: (1) highly sensitive cells, responsible for the large A.C. and D.C. potentials in the endolymph; (2) cells, like those of Hensen and Claudius (and Reissner's membrane somewhat), which do not produce first-order microphonics but act like "biological" D.C. batteries (negative charge inside cell wall, positive charge outside); (3) electrically indifferent tissues, like the basilar membrane ligament, which produce no potentials and act as resistances.

Tasaki [105] uses differential amplifiers to record the electrical responses of the cochlea of the guinea pig. When the acoustic stimuli (pure tones + pips) are kept at a constant sound pressure level, the phase and amplitude characteristics of the

microphonics in the four turns—with respect to a round-window reference electrode—resemble closely the behavior of the Békésy traveling wave. In the basal turn, however, the potential is higher than is indicated by the mechanical deformation of the Békésy wave.

With injections of isotonic KCl solution and shifts in the position of the reference electrode, it is possible to isolate the microphonics from the action potentials for sections of the basilar membrane 1 to 2 mm. long. In general, the voltage of the microphonics increases linearly with sound pressure up to a limit which varies with frequency and the position of the electrode. The higher the frequency, the lower is the intensity of the limit of linearity.

Lissajous' patterns show that discontinuities or "breaks" occur for frequencies at a point on the cochlear partition, having a phase difference of 2π from the basal end. Little change in the space-time pattern of the microphonics occurs when holes are made in the bony portion of the cochlea or when the sound wave is delivered at the third turn. As Békésy showed, the behavior of the traveling wave is attributed to variations in elasticity of the cochlear partition, but the phase difference, not the maximum of deformation, is said to account for the change of frequency with position on the partition.

The complexity of the phase and amplitude factors suggests that any hydrodynamic theory of hearing must take into consideration the "direct driving" of the partition by the sound wave, the existence of surface waves, and transverse elastic waves, which appear in different parts of the cochlea either separately or superimposed on one another.

De Vries [106] points out that the Brownian motion of the drum membrane itself is greater than that produced by the noise due to thermal agitation. The apparent flow of energy is close to the absolute threshold of the ear. He reports that the behavior between the tectorial membrane and the hair cells in models of the cochlea is very much like that between the cupula and the hair cells of the lateral line organ of fish. In the latter, negative potentials are created in the upper side of the epithelium when the hairs are stretched (not compressed) by the gliding motion of the cupula over the epithelium. In the same way, an outward motion of the stapes causes the tectorial membrane to move to the left relative to Corti's organ, stretching the inclined outer hair cells and creating a negative voltage above the tectorial membrane and a positive charge below it. Thus the membrane between the hair and the cell is similar to the membrane surrounding a nerve fiber (negative charge outside, positive inside). The source of the potentials is in the organs that can be stimulated electrically, i. e., in those organs found below the negative charge.

In the inner ear the thermal agitation of the Brownian movement of one cell covers too large an area to interfere with the excitatory electrical charge produced by the combined action of the hair cells during a displacement of the tectorial membrane. Since the tectorial membrane and the hairs are not stiff enough, the transport of energy to the sense cells is not provided by mechanical coupling but by "rigid" electrical coupling.

Rosenblith and his associates [107] state that auditory fatigue is a temporary loss of auditory sensitivity due to previous stimulation. Several types of fatigue are characterized by different time courses, degrees of reversibility, and frequency pat-

terns of impaired response. There has been a great variability in reported data concerning these characteristics. Many efforts have been made to correlate temporary deafness in man with changes in the electrical audiogram in animals. These have been unsuccessful. The electric potentials recorded from electrodes in the cochlea have at least two components: (1) cochlear microphonics, which reproduce fairly accurately the wave form of the acoutic stimulus, and (2) action potentials derived from nerve pathways in the lower auditory tracts. When pure tones are used as stimuli, the microphonic potentials generally obscure the neural components, but if stimuli such as sharp clicks are used, these two can be distinguished in time. After stimulation with a tone of 105 db. in a cat's ear, there is a sizable temporary reduction in the amplitude of the neural component, and the neural response to weak clicks is abolished for a time. During this time, there is no significant alteration in the amplitude of the most prominent microphonics. There is also a parallel between the recovery of the first neural component at the round window and the auditory cortex. The authors suggest the possibility of a relationship between auditory fatigue in the human ear and the magnitude of neural responses in animals.

Watson and Frazier [108] confirm a previous observation that when a pure tone and thermal noise are simultaneously introduced into an ear, there is a rise in pitch of the pure tone. The effect is a function of the levels of the pure tone and thermal noise.

Using 25 subjects with essentailly normal hearing, Wilson [109] exposed them for five minutes to a 2048-cycle tone at an intensity of 80 db. (using a standard audiometer). Thresholds were then made at intervals for 4096 cycles and the fatiguing effect determined. The data indicate that certain ears are more susceptible to auditory fatigue than others, and Wilson feels that these ears should be considered abnormal.

The experimental work in auditory fatigue done in 1943 at Harvard [110] was again published as a supplement to the *Acta oto-laryngologica*. The results are worth consideration. A total of 19 subjects were used; 16 of them had normal hearing, and the other 3 showed evidence of high-tone losses. Auditory fatigue was evaluated, using exposures to pure tones from 500 to 4000 cycles and also intense noise up to 130 db. for times ranging up to about one hour. The subjects were tested not only for threshold levels but also for diplacusis, articulation, and loudness matching.

The author found no evidence of cumulative injurious effects, even though temporary impairment was regularly produced. The greatest hearing loss was found to occur about half an octave above the exposure tone, but with long exposures, the hearing loss extended to all tones above the exposure frequency. Perlman and others found similar effects in their work. Individual susceptibility to auditory fatigue varied greatly, even in the same person. The authors feel that threshold measurements are not adequate measurements of the impairment of auditory function. Alteration of loudness perception and diplacusis are important factors to be investigated. The authors believe that exposure to band spectrum noise, such as in planes, which causes widespread hearing loss that is usually most severe in high frequency ranges, is relatively ineffective in producing diplacusis.

Hood [111] suggests the presence of two different types of auditory fatigue: (1) poststimulatory, which occurs after stimulation, and (2) perstimulatory, or adapta-

tion, which occurs during stimulation. The latter has received little attention. A self-recording and self-operating audiometer of the Békésy type is used to measure fatigue. After testing three normal subjects, Hood concludes that between 90 and 95 db. there exists a critical fatiguing intensity below which fatigue is slight and above which it increases rapidly. He further believes that the pain threshold is not a safe guide to the onset of serious pathological changes in the cochlea. His results indicate that there is no critical duration for a fatiguing tone; it is the intensity rather than the duration which determines the difference between auditory fatigue and stimulation deafness, the former being physiological and the latter pathological.

Hood substantiates the experimental findings of Derbyshire and Davis on equilibration by showing that maximum fatigue occurs when stimuli frequencies of 900, 1800, and 2700 are used. Fatigue ocurs in the end-organ, and a damaged inner ear is more susceptible to fatigue than a normal ear, acording to results obtained from three patients. The duplicity theory of hearing is supported by two experimental findings: (*a*) the lower frequency limit of fatigue remains constant with intensity, while the upper limit becomes progressively higher; (*b*) the frequency distribution of fatigue varies with intensity and extends above the stimulus frequency with high intensities.

Using loudness-balance testing, the author determines perstimulatory fatigue not only of the threshold receptors but other nerve elements acting at higher intensity levels. This type of fatigue reaches its maximum in about three and a half minutes and is independent of the intensity of the stimulus. Its recovery is most rapid in the first 10 seconds and is complete after one minute. The total fatigue in any receptor group of specific size is independent of the intensity of the stimulus. Hood investigated this type of fatigue in 25 patients with unilateral Ménière's disease.

Hallpike and Hood [112] observe a striking resemblance between perstimulatory fatigue (adaptation) characteristics of the normal human ear and the time characteristic phenomena of stretch receptors of frog muscle studied by B. H. C. Matthews. For the ear, maximum adaptation occurs after a period of prolonged stimulation of three and one-half minutes, full recovery occurring after a minimum rest period of one minute.

In a previous study, the authors found that "complete" loudness recruitment and structural changes in the hair cells occurred in all cases of unilateral Ménière's disease but not in cases of eighth-nerve degeneration. Their conclusion was that loudness recruitment arose from the disordered function of the hair cells caused by Ménière's disease.

Continuing their investigations with the Fowler binaural balance test, Hallpike and Hood find that ears affected by Ménière's disease and demonstrating recruitment appear to share with the adapted ear phenomena which are closely akin to on-effect normality and relapse. They suggest, therefore, that loudness recruitment is only one element of a complex derangement of cochlear function due to disease or injury of the hair cells of the organ of Corti, independent of nerve-fiber degeneration.

Using 10 bands of noise (160 to 6600 cycle range) and six pure tones (125 to 4000 cycle range) as fatiguing stimuli, Hirsch and Ward [113] observed a diphasic

recovery curve, i. e., regain of normal threshold about one minute after a three-minute exposure followed by a rise in threshold (bounce) one minute later, for different sounds at intensities ranging from 100 to 120 db., sound pressure level.

The bounce appears for clicks at frequencies below 1000 cps (maximum deviation at 500 cps), for noise and pure tones at frequencies approximately 1000 to 5000 cycles (maximum about 4000 cps.). With a 500-cycle stimulus, the over-all fatigue and bounce are reduced for clicks as the intensity (120 to 100 db.) and duration (six to one and one-half minutes) are reduced. Qualitative differences for clicks (thuds) are noted, which tend to disappear as fatigue approaches zero. Although the recovery curve varies with each observer, it remains consistent for each individual observer.

Facilitation (temporary reduction in absolute threshrold below normal value) was observed for clicks when the fatiguing tone and test tone were the same value. After stimulation by a pure tone, the residual fatigue after eight minutes tends to be the same at all frequencies for noise and pure tones. After stimulation by noise, however, fatigue after eight minutes seems to increase as the frequency of the test tone increases. A second bounce occurs consistently five or six minutes after stimulation in half the cases, indicating that the phenomenon may be oscillatory. It is interesting to note that Hallpike and Hood in the previous study—using a 1000-cycle tone but a lower intensity (80 db.)—make no mention of having encountered the bounce phenomenon.

Pollack [114] finds that the absolute threshold of white noise (of constant sound time fraction) increases as the frequency of interruption is increased, i. e., as the interval between successive noise bursts decreases. As this interval is increased, the burst level at threshold is decreased. To maintain constant loudness, the burst level of an interrupted noise must be increased as the rate of interruption increases, and if the level of interrupted noise is held constant, the loudness of the noise decreases as the rate of interruption increases. Unlike that for continuous noise, the masking effectiveness is increased with an increase in the rate of interruption of white noise. In many cases, the burst level of an interrupted white noise (sound time fraction of 0.45) is less than the burst level of an equally loud continuous noise, i. e., with the same burst amplitude (and less energy) the interrupted noise sounds louder.

Maximal effectiveness is found in the region 2 to 10 interruptions per second. Evidence exists where increase of sensory function for other phenomena occurs in this region of interrupted frequency, e. g., brilliance of interrupted light (10 per second) and intensity discrimination between two tones of different frequency (3 per second).

It is suggested that the occipital alpha brain potential (fundamental frequency about 10 per second) might be related to this matter. The alpha rhythm may represent the electrical output of a process acting as a scanning mechanism of the entire cortex. To be maximally effective, bursts of excitation must coincide in time with critical durations limited by successive sweeps of the alpha scanning mechanism.

In a concomitant study, Pollack [115] attempts to determine the differential threshold between the level of repeated bursts of noise and the level of noise (increased or lowered at will) in the interval between these bursts of noise. For intervals greater than 55 milliseconds the just detectable decrement is a constant

and is indepedent of the duration of the interval. Below this "critical duration" the just detectable decrement increases proportionally as the interval decreases.

Studies by other investigators show that the duration of persistence of auditory stimulation for white noise is about 55 milliseconds. Then the auditory effect experienced from any burst will tend to be affected by a previous burst if the interval between them is less than the critical duration. For such cases a larger differential in intensity will be required to detect a decrement in intensity, the magnitude of this differential being proportional to the level of persistence.

The investigation of the differential sensitivity of the ear as a function of the interval between bursts of white noise suggests a procedure for the indirect measurement of auditory persistence.

Pollack [116] finds that the best estimate of the loudness function for white noise is represented by a mean scale determined from a "direct" scale based on monaural-binaural loudness ratio determinations with white noise and a "derived" scale based on extrapolation from the Fletcher loudness scale of a 1000-cycle pure tone. The new scale is a compromise scale computed from the ordinate values of the direct and derived scales in which loudness in tones is plotted as a function of the sound pressure level of white noise in decibels.

The loudness ratio determinations for the 1000-cycle tone and white noise are obtained over a wide intensity range for monaural listening by instructing subjects to adjust the intensity of a variable signal to sound half as loud or twice as loud as a standard signal. After a preliminary right-ear versus left-ear loudness match, the intensity of a binaural signal is adjusted to obtain the monaural-binaural loudness equation. Noise versus tone (heterophonic) loudness matches are determined by adjusting the intensity of a 1000-cycle tone to sound equal in loudness to a white-noise signal.

A supplementary investigation (to be published later) reveals discrepancies between results found for white noise and the loudness of speech. These differences may be due to perceptual constancies built up to speech with the passage of time.

Pollack next uses his loudness-ratio data to determine the relative loudness of noise bands over a frequency range 60 to 5800 cps. The sound pressure level of a narrow band of noise (constant in mels) is more nearly proportional to the level of an equally loud 1000-cycle tone than to an equally loud wide-band noise. At very high and low sound levels, frequencies at the center of the audible noise spectrum make a greater contribution to loudness than do either low or high frequencies. At a fixed over-all sound pressure level, noise sounds louder when it is spread over the spectrum than when concentrated in a narrow range of upper and lower frequencies, even when noise frequencies to which the cat is less sensitive are added.

Not at intermediate levels, but at threshold and very high levels is it effective to concentrate the noise spectrum over a narrow frequency range. Pollack assumes that at threshold the displacement density on a small area of the basilar membrane is nearly proportional to the concentrated spectral density of a narrow band of noise. At intermediate levels, the band must be widened to saturate a greater area of critical excitation. At the highest levels, the "spread" of displacement may be great enough so that both narrow and wide bands of noise operate effectively over the same saturated region of response.

Garner [118] used 18 different observers to make half-loudness judgments of a fixed intensity tone in 10-db. steps from 10 to 100 db. re 2×10^{-4} dynes per cm. Regardless of the intensity of the fixed tone, the amount of attenuation varies with the observer. Observers differ least at low intensities and most at high intensities. However, for any one observer the variability is greatest at low intensities and least at high.

Even after a lapse of several days, the subjects remain consistent in their judgments. It appears that an observer can establish a criterion for what he thinks is half loudness, i. e., he is consistent in his choice of a particular fraction, but he is not sure that his choice is half as loud. In a sense, fractional judgments of loudness are reliable but not valid.

Considerable disagreement exists between these data and those of previous investigators. Moreover, a loudness scale in tones computed from the loudness unit data of the American Standards Association show a lack of linear relationship with a similar scale computed for this experiment.

The analysis of variance for half-loudness judgments in the present study reveals that an observer's judgments are influenced by the initial difference in intensity between two tones, the range of the stimulus intensities heard, and the type of judgment required. Although none of the observers was so experienced, Garner points out that familiarity with acoustic and electrical measurements also tends to influence one's judgments.

Guelke and Helm [119] challenge the validity of the Churcher-King extrapolated zero-phon loudness contour for low frequencies. Having determined the threshold of audibility for 38 subjects binaurally (free field) and for 10 subjects monaurally (earphone), they find that at 100 cycles an increase of 14.5 db. above the threshold is necessary to match the loudness of a 1000-cycle tone 10 db. above threshold. However, to achieve equal loudness with a 1000-cycle tone whose intensity is increased from 10 to 20 db. above threshold, a 100-cycle tone requires only an 8-db. rise.

The equal loudness charts show that at low frequencies (100 cycles and below) the contours tend to rise and crowd together in the intensity range approximately 35 to 85 db. above the reference level. But the present study finds that the zero-phon contour at these low frequencies does not behave in the same way. Some explanation for this anomalous deviation may lie in the experimental evidence which shows that contractions of the intra-aural muscles (such as the tensor tympani) increase the sensitivity of the ear at high frequencies but decrease the sensitivity at low frequencies.

Thus when a loudness comparison is carried on between a 1000-cycle tone and a 100-cycle tone, the tension of the muscles would discriminate against the low frequency note. But when the threshold of only low frequency notes is being measured, the muscle tension is less resulting in a threshold curve which is considerably flatter than the extrapolated zero-phon curve.

Pollack [120] asked 33 observers at what loudness level they would prefer to listen to pure tones (125 to 8000 cps in octave steps) against no noise and white noise backgrounds over a large intensity range. The most comfortable listening level (MCL) contour determined from monaural and binaural listening with no noise

resembles the equal loudness contours of the middle intensity range. When compared with the average monaural MCL level, the average binaural MCL level at each of both ears is significantly lower. The upper and lower limits of the range of comfortable listening levels at various frequencies show on the average the greatest range for middle frequencies, the least for lower, and intermediate for the higher frequencies. The range of comfortable listening levels varies from about 20 db. at the lowest frequencies to 35 db. at middle frequencies. The variability (standard deviation) increases among observers of comfortable listening levels as the frequency is increased.

As the background is filled with noise, both the MCL and the upper and, especially, lower limits of the range of comfortable listening levels are increased, but the range itself is decreased. The data show that for a given listening level, observers can stand louder signals better in high-noise than in low-noise backgrounds.

The decrease in the range of comfortable listening levels for listeners with high-tone deficiencies (nerve type deafness) resembles closely that for normal observers listening against a background of noise. This suggests that the MCL level might be used as a gross diagnostic tool in evaluating nerve-type and conductive-type deafness. A conductive-type patient would show an increase in the upper and lower limits of the comfortable listening levels but with a "normal" range, whereas a nerve-type patient would exhibit a normal upper limit, a greatly increased lower limit, and thereby a significantly contracted range of comfortable listening levels.

Investigators report that for low speech to noise (S/N) ratios greater intelligibility is obtained by clipping the peaks of high amplitude speech waves and amplifying the entire signal to peak capacity. Pollack [121] attempts to discover if the frequency band of a speech signal is responsible for the high intelligibility peak of clipped speech at low S/N ratios and of unclipped speech at high S/N ratios.

A comparison of the gain functions (articulation score parameters) for different frequency bands obtained with low-pass and high-pass filters shows only slight differences between clipped and unclipped speech. He concludes that for a given frequency band, the intelligibility of unclipped speech relative to infinitely clipped speech varies as the S/N ratio and is independent of the frequency band employed.

Without suggesting what to listen for, Garner and Wertheimer [122] asked 54 "normal" listeners whether two successive tones with the same phase lead sounded the same or different when one was introduced in one ear and the second in the other ear. With two intensities (50 and 90 db.) for eight frequencies in the range 200 to 2000 cps, the observers judged the phase leads at 0, 30, 90, 120, 150, and 180 degrees.

For each frequency the maximum heard differences occur with a phase lead of 90 degrees. As the lead changes toward 0 or 180 degrees, the percentage of heard differences decreases. At 2000 cycles observers report very little difference between the tones. But as the frequency is decreased, a considerable difference is heard at each phase lead, the difference becoming greater as 200 cycles is reached.

The interaural phase difference thresholds computed from the data show that at any audible intensity the time difference between the intensity maxima of the pure tones of the two ears is independent of frequency—up to the critical frequency where differences in phase are not perceived.

The perceptual effects of phase differences are such that all observers identify the differences as localization. However, 29% place the effect of the sound source in space or in the head, whereas 71% reported it as a change of stimulus from one earphone to the other. Fully 76% noted an accompanying change in pitch and 22% a change in loudness. Other effects, such as volume, richness, heaviness, etc., were also reported by 57% of the observers.

Morgan and associates [123] attempt to restudy the relation of pitch (as a monotonic function of frequency) to intensity. The investigators find good agreement between the Snow method and their "ladder" method, which compares a pure tone to a standard tone always 10 db. below the compared tone, in 10-db. steps (starting from 100 db. re 2×10^{-4} dynes per cm.^{2}), for seven frequencies in the range 125 to 8000 cps.

The data between individual observers show a lack of consistency, no two sets of data being alike. With increasing intensity, the functions for one ear might go up, and for another ear they might go down. Three statistical parameters (the median, the first quartile, and the third quartile points) of a "population of ears" show that typical ears do not change more than 2% in pitch with increasing intensity, whereas atypical ears manifest a good deal of change with intensity. At low frequencies the pitch falls with intensity; at high frequencies (4000 and 8000 cps) the pitch rises. This trend is also true for typical ears, but the change is slight. Also, the pitch for the latter tends to rise slightly and then decrease with rising intensity in the intermediate frequency regions (1000 and 2000 cps).

Over a long period of time, Munson and Wiener [124] have been trying to determine why the ear is less sensitive to a pure tone (4 to 10 db. loudness loss) when the sound is heard from earphones than when the ear receives the stimulus from a loud speaker.

This condition exists for frequencies below 300 cps at zero-loudness level and at a level of 60 to 80 phons. Of all the hypothesis proposed to account for the loss, none seems to provide a satisfactory explanation. The static pressure built up in the ear by a snug earphone is not great enough to account for the "missing decibels." Nor is a solution found in the idea that the middle-ear muscles are contracted by pinching the pinna or the sensitive walls of the canals, resulting thereby in a reduction of ear sensitivity at low frequencies. When the earphones fit the ears snugly —but a tone from a loud speaker is permitted to leak in under the receivers—the ear pressures for equality of loudness from the phones are the same as when the stimulus comes from the loud speaker with the phones removed. Measuring the sound pressure in the ear canal from earphones at low frequencies is not a reliable criterion of the magnitude of the sensation experienced by a listener. The authors hope their findings may stimulate an interest in the problem.

Webster and his associates [125] investigate the masking properties of thermal noises with abruptly changing spectra and the pitch shifts of pure tones near the abrupt changes. The comparison of masking inside and outside of a gap cut from a noise spectrum is somewhat better with a band shaped like a resonance curve than with the band of the well-known critical band hypothesis.

With noise of uniform spectrum (flat noise) as a background, the threshold of a pure tone is raised. With gapped spectrum noise, the masked threshold of a pure

tone at the edges of the gap is like that for flat noise. In the middle of the gap it shifts to about 25 db. lower. The thermal noise with an octave gap filtered out can be used to mask-out distracting noise outside the gap.

White noise raises the pitch of a pure tone, and the amount of rise can vary from nothing at 500 cycles to over 100 cps (3/4 semitone) at 3000 cycles. If the pure tone finds itself in the upper half of the gap of an abruptly sloped noise spectrum, no rise in pitch occurs. But in the lower half of the gap, the pitch of the tone is higher than it would be with a background of flat noise.

The amount of masking affected by high-passed, low-passed, uniform, and gapped noise bears no simple relationship to the amount of pitch shift. Nor does the decrement in loudness bear a simple relationship to changes in pitch. The data seem to indicate that the pitch of pure tones masked by abruptly changing noise spectra shifts from the locus on the basilar membrane bearing the noise components.

The problems of communication are, of course, basic to audiology. Otologists would do well to digest the lucid discussion of "Information Theory" by Weaver.[126] The nature of selective information as it applies to communication theory is clearly explained by analogies of complex thoughts to everyday experiences. This is a practical article and opens appealing avenues of thought. Quantitative values for information are derived and units such as a "bit" clearly defined. Beginning with a brief explanation of the "binary" number system, the author easily leads into the nature of selective information, showing that the more unexpectedness in a communication the more information it contains. He discusses the probability of selecting messages and how the probability of selecting various messages is dependent on past selections, the so-called "Markoff Process." He further explains how this is still in keeping with one's freedom of selection of messages and making one's own decisions. The idea of redundancy is also described as well as a precise manner of measuring it. The entire presentation is most stimulating and important to an open mind. Peterson [127] applies the information theory to experimental phonetics and describes the steps required in designing voice-operated devices.

Davis [128] completes the series on the information theory by demonstrating its application to research in hearing. He discusses the experiment by Miller, Heise, and Lichten [129] to determine articulation functions for word lists against a background of noise. The intelligibility context of the words were varied and the functions of the various types of test material compared. A definition for an "intelligible" speech unit is given in conformance with the information theory. The authors show that by simply restricting the vocabulary the intelligibility of the average word is increased by 18 db. It is also demonstrable that communication can be much more improved by standardizing procedures and vocabulary than by mere repetition one or two times. Davis emphasizes the influence that information theory has on experimental work in hearing.

Many other notable contributions not herein reviewed have been made to the literature in audiology. Among these are an excellent bibliography on hearing by Kryter and publications by the Committees on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology. The primary purpose of this short review has been to integrate the many disciplines which contribute to the expanding field of audiology.

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Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF JUNE 1, 1954

Acousticon Models A-17, A-180, & A-185
Mfr., Dictograph Products Inc.
95-25 149th St.
Jamaica 35, N. Y.

Audiotone Models 11 & 15
Mfr., Audio Company of America
5305 N. 6th St.
Phoenix, Ariz.

Audivox Models Super 67 & 70
Mfr., Audivox, Inc.
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"
Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Cleartone Model 700
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Junior Model D-2

Dahlberg Model D-3 (Tru-Sonic)

Dahlberg Model D-4 (Tru-Sonic)
Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Fortiphone Models 19LR, 20A, 21C, & 22
Mfr., Fortiphone Limited
Fortiphone House
247 Regent St.
London, W.1, England

Distr., Anton Heilman
75 Madison Ave.
New York 17

Gem Model V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corp.
708 W. 40th St.
Minneapolis 8

Maico Maxitone

Maico Model J

Maico Top Secret Model L
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Micronic Model 303

Micronic Model Mercury
Mfr., Audivox, Inc—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Models T-10 & T-612
Mfr., The Microtone Corporation
758 S. Mississippi River Blvd.
St. Paul 1

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C

Normatone Model D-53
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Models B-15 & B-30

Otarion Models F-1 & F-3

Otarion Model G-3

Otarion Model H-1, Custom "5"
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Model 62 Starlet

Radioear Model 72

Radioear Model 82 (Zephyr)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16

Distr., Radioear Corporation
306 Beverly Rd., Mt. Lebanon
Pittsburgh 16

Silvertone Models H-16, J-92, & P-15
Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck & Co.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 910, 920, 925, 940, 966, 977, & 988
Mfr., Sonotone Corporation
Elmsford, N. Y.

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 99, 200, 300-B, 400, 500, 952, 953, & 1700
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model
Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Zenith "Regent"
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All aforementioned hearing aids have vacuum tubes.)
Accepted hearing aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED

Acousticon Model A-300	1 transistor and 2 tubes
Acousticon Model A-310	1 transistor and 2 tubes
Acousticon Model A-330	3 transistors
Acousticon Model A-335 Mfr., Dictograph Products Inc. 95-25 149th St. Jamaica 35, N. Y.	3 transistors
Audivox Model 71 Mfr., Audivox, Inc. 123 Worcester St. Boston 18	3 transistors
Beltone Concerto Model Mfr., Beltone Hearing Aid Co. 2900 W. 36th St. Chicago 32	3 transistors
Maico Transist-Ear Model O Mfr., The Maico Company, Inc. 21 N. 3rd St. Minneapolis 1	3 transistors
Micronic "All American" Hearing Aid Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division 123 Worcester St. Boston 18	3 transistors
Microtone Model T1 (Red Dot)	3 transistors
Microtone Model T1 (Yellow Dot)	3 transistors
Microtone Model T31 (Micro-Mite) Mfr., The Microtone Corporation 758 S. Mississippi River Blvd. St. Paul 1	1 transistor and 2 tubes
Otarion Model C-15	1 transistor and 2 tubes
Otarion Model D-1	3 transistors
Otarion Model F-22 Mfr., Otarion, Inc. 4757 N. Ravenswood Ave. Chicago 40	1 transistor and 2 tubes
Radioear Model 820 Mfr., E. A. Myers & Sons, Inc. 306 Beverly Rd. Mt. Lebanon Pittsburgh 16 Distr., Radioear Corporation	3 transistors
Silvertone Model H-25 Mfr., The Dahlberg Co. Golden Valley Minneapolis 22	3 transistors

Distr., Sears, Roebuck & Co.
925 S. Homan Ave.
Chicago 7

Sonotone Model 1010 — 1 transistor and 2 tubes

Sonotone Model 1111 — 3 transistors
Mfr., Sonotone Corporation
Elmsford, N. Y.

Telex Model 954 — 1 transistor and 2 tubes

Telex Model 956 — 3 transistors
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Unex Model TR-3D — 3 transistors
Mfr., Nichols & Clark
Hathorne, Mass.

Zenith Model Royal-T — 3 transistors

Zenith Model Super Royal-T — 3 transistors
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

Semiportable Hearing Aids Accepted

Ambco Hearing Amplifier (Table Model)
Mfr., A. M. Brooks Co.
1222 W. Washington Blvd.
Los Angeles 7

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Model Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

ROENTGEN DEMONSTRATION OF THE SEMICIRCULAR CANALS IN PAGET'S DISEASE. K. KETTUNEN, Am. J. Roentgenol. **70**:564 (Oct.) 1953.

In 1950 Newman and Rechtschaffen reported a case of Paget's disease involving the skull in which there was roentgenologic visualization of the semicircular canals. Kettunen adds another case in which roentgenograms of the skull clearly visualize the semicircular canals on both sides in a patient with typical changes of Paget's disease in the skull and in other bones. It is probable that the visualization of the semicircular canals in Paget's disease of the skull is caused by a combination of osteosclerosis of the osseous labyrinths of the inner ears with osteoporosis of the temporal bones. Impairment of function of the inner ear is a characteristic finding in Pagets disease complicated by deafness.

WEILAND, Grove City, Pa. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

CHRONIC OTORRHEA. L. M. SELLERS, Eye, Ear, Nose & Throat Month. **32**:495 (Sept.) 1953.

Chronic otorrhea and its underlying pathologic cause, chronic tympanomastoiditis, is an important problem of the otologist. Chronic tympanomastoiditis is an infection of the tympanum and its adnexa in which spontaneous repair has been attempted and partially attained but has been prevented from reaching completion. If there is no attempt or an inadequate attempt at arrest and repair, the case progresses rapidly to acute suppurative mastoiditis with its complications. Chronic tympanomastoiditis with its chronic otorrhea is an end-product of acute suppurative otitis media rather than of acute suppurative mastoiditis. Treatment is determined by the areas involved and the degree of their involvement. Both the patient and the physician should understand that the objective to be attained is not the cessation of a discharge. It is the elimination of an actual or potentially dangerous focus of active infection.

There are two general classes of treatment: nonsurgical or minor surgical and surgical. Medical, or nonsurgical, treatment may be the method of choice, but the limits of medical or minor surgical treatment must be realized, and the cases lying beyond these limits must be recognized and treated promptly with the operation that is indicated. It is an erroneous concept that the modified atticoantromastoidectomy or the complete tympanomastoidectomy is but a further step of the so-called "simple" mastoidectomy. This is a fundamental error, as the operation has for its purpose the completion of an aborted or perverted process of repair having its start and, in most cases, its principal focus in the attic and antrum. It is at this point that surgery should begin, and it is from this point that it should progress in all directions until the work has been completed.

For seven years Sellers has used the endaural approach exclusively and has found no case in which it was not superior to the postauricular approach. It provides the most direct visual and surgical approach to all portions of the field. The antrum and attic are exposed initially, and all subsequent procedures are directed under vision away from danger zones.

JENNES, Waterbury, Conn.

AERO-OTITIS: ETIOLOGIC AND THERAPEUTIC CONSIDERATIONS. B. C. TROWBRIDGE, Eye, Ear, Nose & Throat Month. **32**:500 (Sept.) 1953.

Aero-otitis results from the inability of the Eustachian tube to adapt to the rapidly increasing atmospheric pressure during descent while flying. This results in the accumulation of sterile fluid in the middle ear, with its frequently characteristic amber color. There may be visible air bubbles and a curved fluid line, which may move with motion of the head.

With the usual palliative treatment of the acute symptoms by local therapy of the nose and throat with inflation of the ears, the fluid in the middle ear usually disappears within 7 to 21 days.

Aspiration tympanotomy is the most effective way of relieving the symptoms and shortening the convalescence. This may be accomplished with minimal discomfort without anesthesia or with the use of local anesthesia, consisting of a 10% solution of cocaine in aniline oil, applied topically to the membrana tympani. Trowbridge utilizes his ear aspirator, which he has previously described in the literature and which simplifies the procedure.

JENNES, Waterbury, Conn.

A SIMPLIFIED METHOD OF PATCHING TRAUMATIC PERFORATIONS OF THE TYMPANIC MEMBRANE. C. D. BENTON JR., Eye, Ear, Nose & Throat Month. **32**:633 (Nov.) 1953.

Benton reports the successful closure of nine cases of traumatic perforations of the tympanic membrane with cigarette-paper discs. The edges of the perforation, if smooth, are touched with trichloroacetic acid. Tincture of benzalkonium (Zephiran) chloride is applied to the ear drum and to the paper disc. Benton describes a device which he has employed for transferring the paper disc to the ear drum. The ear drum, with patch in situ, is then dusted lightly with oxytetracycline (Terramycin) and boric acid powder. Healing generally took place within 14 to 45 days, without any additional treatment. In two cases, when the patch was removed, it was found that the closure was incomplete. A fresh paper patch was reapplied to the drum and left in place until healing was complete.

JENNES, Waterbury, Conn.

OTITIS EXTERNA: THE FACTS OF THE MATTER. J. W. MCLAURIN, J. A. M. A. **154**:207 (Jan. 16) 1954.

McLaurin outlines the important principles that should guide the otologist in the management of the manifold cases of otitis externa that are seen in practice. Among many interesting discussions he quotes Fabricant, who stated some time ago that "the guiding plan should be a maximum of inspection and a minimum of therapy." To this McLaurin adds the utmost importance of the most scrupulous care in cleansing the ear and keeping it clean and dry.

Cultural studies of the ear were made in 157 cases, and numerous sensitivity tests were made with the current chemical agents used.

A large proportion of the cases studied indicated that there was no clear causative factor. The most important consideration of classification is the distinction between bacterial otitis externa and diseases of fungous origin. All therapy should be conducted on a realistic basis, and any specific treatment should be applied according to precise principles.

GORDON, Philadelphia.

CAUSES OF FAILURE IN SURGICAL FENESTRATION: A FOLLOW-UP OF 3,000 CONSECUTIVE CASES. GEORGE E. SHAMBAUGH JR., J. Internat. Coll. Surgeons **19**:104 (Jan.) 1953.

Continuous study and observation brings to light certain techniques that have largely overcome some of the failures of fenestration surgery. The continuous irrigation technique to create a fenestra free from bone dust has done much to lessen the likelihood of osteogenic closure. Good enchondralization, so that the skin flap will fit tightly against the fenestral margins as healing and contraction occur, and preservation of the intact endosteal membrane within the labyrinth to the knife-like edges of the fenestra will aid in optimal healing and preservation of the created fenestra.

The strictest possible aspetic technique during the operation and for a postoperative period of six weeks must be observed. Adequate preoperative preparations must be observed. The otolaryngologist, working in infected areas, is not likely to realize what diligent aseptic procedures are necessary in fenestration surgery.

Two major problems that await solution are the further reduction of the incidence of postoperative serous labyrinthitis and a method of eliminating the 25 db. of residual conductive loss after successful fenestration that still prevents the restoration of entirely normal hearing in patients with normal function of the cochlear nerve.

GORDON, Philadelphia.

TUBERCULOUS OTITIS MEDIA. LINDEN H. WALLNER, Laryngoscope **63**:1058 (Nov.) 1953.

This is a well-written article on tuberculosis of the middle ear. It is always secondary to another focus, usually the lungs. There is a characteristically insidious onset, with painless discharge. The incidence is not high, in this study being one-quarter that of laryngeal tuberculosis. There was early and often severe hearing loss of a conductive nature. Mastoid x-rays showed minimal changes. Serious complications were rare, a finding contradictory to most writers.

HITSCHLER, Philadelphia.

SYMPOSIUM ON HEARING TESTS AND THEIR SHORTCOMINGS. P. F. KING, M. R. DIX, J. D. HOOD, and R. S. DADSON, Proc. Roy. Soc. Med. **46**:991 (Dec.) 1953.

At the meeting of the Otology Section of the Royal Society of Medicine on Feb. 6, 1953, Air Vice-Marshal E. D. D. Dickson presided. Wing Commander King spoke on "Some Imperfections of the Free-Field Voice Tests." Conversational voice should be heard at 20 ft. (6 m.) by the normal ear, but various examiners report different readings. This figure is therefore unreliable. Whispered voice does not depend on vibrations of the vocal cords but is produced by the residual air in the lungs after expiration. Whispered sibilants are heard further than spoken low-tone words. The weaknesses of the test for the whispered voice are failure to standardize the technique; inability to control the pitch and intensity of a whisper; lack of control of "ambient tones." Lack of control of the latter seems common. Tests should be conducted in a quiet room, "sound-damped, having an ambient noise level of 50 db. or less." Test rooms vary in acoustics. Any change in the contents of a room (furniture) changes its transmission characteristics. Reference is made to the Committee on Hearing of the National Research Council, which in 1948 adopted pure-tone audiometry. "In the future, speech audiometry will probably be used as the basis for hearing standards, and this will be the ideal."

Drs. Dix and Hood, of the National Hospital, Queen Square, London, described "Modern Developments in Pure Tone Audiometry and Their Application to the Clinical Diagnosis of End-Organ Deafness." They state: "There is, at the moment, no degree of standardization in the manner in which the onset of the tone is controlled in any of the commercial audiometers and they appear to vary greatly."

Mr. R. S. Dadson discoursed on "Some Possible Sources of Error in Audiometry and Their Remedies." He spoke of the physical limitations, the importance of the "reference threshold," and the lack of adequate calibration facilities. In the past there has been no agreement as to the threshold values at different frequencies. Undesirable effects are found in the variability of the coupling between the earphone and the subject's ear. Leakage of sound results from imperfect sealing between the earcap and the ear. In order to reduce errors the designs of earphones for use with audiometers deserves further study. There are limitations due to bone-conducted sounds and "the mechanical impedance of the mastoid process."

The work done to date has been a part of the research program of the National Physical Laboratory (England).

ABSTRACTOR'S COMMENT: When one considers the years that have gone by and the number of investigators in physical laboratories, it seems about time that some more definite conclusions should be "openly arrived at" and that better standardization, both here and abroad, be established. It may be that recent work on transistors shall bring about a better understanding of aural functions and disabilities.

I. W. VOORHEES, New York.

Pharynx

RELATIONSHIP OF TONSILLECTOMY TO POLIOMYELITIS. T. C. GALLOWAY, J. A. M. A. **151**:1180 (April 4) 1953.

Reports from various centers have led to conclusions that are difficult to evaluate. Experimental and clinical data have not clarified the real relationship of tonsillectomy to poliomyelitis.

Galloway has diligently searched for statistics and has given much thought to the evaluation thereof.

From his study of the evidence, he concludes: "Tonsillectomy does significantly increase the incidence of bulbar poliomyelitis when done during epidemics. The over-all incidence is probably

increased but perhaps only by the increase of bulbar poliomyelitis. Elective tonsillectomies should be restricted in epidemic periods. Investigation should be made of a similar relationship of dental and other oral and general operations to poliomyelitis and the need of restricting them in epidemic."

GORDON, Philadelphia.

ENDOTRACHEAL ANESTHESIA FOR TONSILLECTOMY AND ADENOIDECTOMY IN CHILDREN. J. W. PENDER and O. E. HALLBERG, J. A. M. A. **153**:1073 (Nov. 21) 1953.

Pender and Hallberg list the advantages and disadvantages of endotracheal anesthesia with a number of general rules for avoiding trauma during intubation as well as avoiding motion of the tube in the larynx and the utmost importance of using chemically and bacteriologically clean tubes.

They support their views and data with a listing of three tables indicating the exact size of the endotracheal tube with respect to the age of the child (989 cases), the size with respect to the weight of the child (602 cases), and the duration of intubation (1,023 cases).

They conclude that disadvantages of this type of anesthesia have been overemphasized and that the working space of the surgeon is not obstructed, in fact, visualization is improved.

GORDON, Philadelphia.

TECHNIQUE OF REMOVAL OF ADENOID TISSUE OF THE NASOPHARYNX IN CHILDREN. PHILIP E. MELTZER, J. A. M. A. **154**:228 (Jan. 16) 1954.

Meltzer describes a technique for the removal of adenoid tissue of the nasopharynx which has given him the best results in his many years of surgery. It is performed with the patient in an upright position, under endotracheal anesthesia, with the nasopharynx exposed to the view of the otolaryngologist, so that the adenoidectomy is accomplished under direct visualization. Thus no longer will a "blind" adenoidectomy be performed.

For a fuller description of the technique, the reader is urged to consult the original article.

GORDON, Philadelphia.

Larynx

SPEECH WITHOUT VOCAL CORDS. Editorial, J. A. M. A. **151**:560 (Feb. 14) 1953.

After laryngectomy, it is of the utmost importance that the patient be trained in the production of pseudovoice so that he may be gainfully employed and a socially acceptable member of society.

Artificial mechanical devices may be reserved for the few patients who cannot master esophageal speech. Most patients are able to master esophageal speech within a month or two.

In esophageal speech, the air pocketed in the upper esophagus is expelled or eructed upward through the closely approximated membranous surfaces of the cricopharyngeal sphincter (pseudoglottis) into the hypopharynx. Sound thus produced is converted into speech by the lips, teeth, tongue, and palate. McCall has pointed out that esophageal speech can be mastered if the patient has learned the technique of sustained belching. He should train himself to do so prior (a week to 10 days) to laryngectomy.

There are two types of devices in current use for those patients unable to use their esophageal speech, but many patients have found these mechanical methods objectionable. Persistent practice on the patient's own anatomic and physiological resources is superior to man's inventive genius.

GORDON, Philadelphia.

CARCINOMA OF THE ESOPHAGUS. ALLEN STRANAHAN, WALTER G. GOBBEL JR., and KENNETH B. OLSON, New York J. Med. **54**:519 (Feb. 15) 1954.

Sixty-four cases of cancer of the esophagus were seen by Stranahan and his associates at the Albany Hospital between 1941 and 1950. There were 63 white patients and 1 Negro, aged 50 or over, with the male patients predominating. Of the 23 who used alcohol, 20 were moderate to heavy drinkers. Twenty out of 23 were smokers. Chewing tobacco was not mentioned. Delayed

diagnosis was the rule, and usually about three months intervened from early symptoms to determined cause. Dysphagia was one of the first and most persistent symptoms, followed by substernal pain and cough. The diagnosis was made with esophagoscopy and biopsy in over 86%. Radiograms were helpful but too often misinterpreted. Although the disease process is often regarded as slow-going, surgery was often done too late, and the survival rate was only from five to seven months after operation. Roentgen-ray treatments seem to have had little influence, since those so treated survived 5.2 months compared with those with no such treatment, who survived 6.3 months!

ABSTRACTOR'S COMMENT: Apparently all patients with dysphagia which lasts more than a few days should have an esophagoscopy with biopsy. Temporizing brings defeat. Although surgical resection is prolonged and difficult, death on the table is rare—1 in 64 for this report. Once again, early diagnosis and prompt operation prolong life. The word "cure" is not being used by most authorities in this field.

I. W. VOORHEES, New York.

A METHOD OF ESOPHAGRAPHY IN ESOPHAGEAL CONDITIONS. GIUSEPPE PESCE and LUIGI PARODI, Minerva otorinolaring. **1**:41 (June) 1951.

With a slender esophageal sound provided with a light source consisting of three bulbs, each one connected to a proper system for graphical registration, Pesce and Parodi have completed a study of esophageal peristalsis in the right proportions at three diverse levels and precisely at the average or mean part of the cervical esophagus, middle thoracic esophagus, and the epicardial portion of the esophagus. They have applied this method of investigation by the examination of 37 patients with esophageal conditions, such as esophageal enlargement, herniation of the esophageal hiatus, carcinoma, cicatricial stenosis, esophageal dyskinesia, chronic esophagitis, and have drawn out precise data about esophageal tone and peristalsis in the diverse medical conditions studied.

The information gathered by means of an esophagram not only contributes to accurate knowledge of the physiology and pathology of the esophagus but likewise can aid in a practical fashion as a means of diagnostic interest.

DELUCA, Philadelphia.

Nose

SURGICAL MANAGEMENT OF MUCOCELES OF THE FRONTAL SINUS. M. J. TAMARI and S. H. BEAR, J. Internat. Coll. Surgeons **20**:269 (Sept.) 1953.

Tamari and Bear present a review of the literature on mucocele of the frontal sinus and discuss the various surgical techniques employed in the treatment of this lesion. They review a series of 20 cases in which operation was performed by the external frontal approach, reporting 5 cases in detail.

Preference is for the radical external frontal approach instead of the intranasal, since the best visualization and removal of the cyst, as well as coexistent pathology, can be obtained.

One case showed the superior oblique muscle attached to the thickened wall of the cyst, with erosion of bone around the trochlear fovea, and in this case Tamari and Bear suggested separating as far as possible the attachment of the muscle from the cystic wall and transfixing the remaining attachment with nonabsorbable (silk) or chromic absorbable surgical (gut) sutures into the periosteum or the edge of the orbicularis muscle. In their experience this procedure has completely restored muscular function.

GORDON, Philadelphia.

THE SPHENOID SINUS FROM A SURGICAL STANDPOINT. GEORGE L. WHELAN, J. Internat. Coll. Surgeons **20**:612 (Nov.) 1953.

Whelan has had a great deal of experience in sphenoid sinus surgery. He reviews the surgical anatomy of this sinus and some of the commoner complications of suppuration of the sphenoid sinus. He agrees with A. W. Proetz's dictum against destroying the ostium when the sphenoid sinus is entered, that a perforating instrument or a small curet introduced close to the nasal septum is in a safe area and easy to manipulate, and that this approach to the anterior wall is not too difficult.

Sphenoidotomy is done for the purpose of instituting drainage and ventilation for chronic suppurative infection, yet it must be remembered that excessive ventilation of the sphenoid may cause dryness and discomfort.

Whelan reminds us that the sphenoid sinus ought to be more frequently considered, especially in the ocular cases.

GORDON, Philadelphia.

Miscellaneous

MANDIBULOFACIAL DYSOSTOSIS. P. HURWITZ, A. M. A. Arch. Ophth. **51**:69 (Jan.) 1954.

Mandibulofacial dysostosis is a rare congenital malformation of the facial bones, first described in 1889 by Berry. At that time it also became known as the "Treacher Collins syndrome." There are five forms of the disease: (1) complete; (2) incomplete, in which the deformity is less marked, the ears may be almost normal, but deafness is usually present; (3) abortive, in which only the lid anomalies exist; (4) unilateral, with hypoplasia of the muscles of the face and other symptoms on one side, and (5) atypical.

The palpebral fissures are involved; the malar bones and the mandibles show hypoplasia; malformation of the external ear, and occasionally of the middle and inner ear, is found; high palate and irregular disposition of the teeth, fistulae between the angles of the mouth and the ears, atypical hair growths, facial clefts, and other anatomical deformities occur. Additional features include mental retardation, harelip, clubfoot, and synostosis of the joints. The less salient features are ocular, nasal, oral, aural, and other bony defects of the skull.

The hereditary aspect of the condition has been proved by its appearance in several generations. It seems to have an irreguiar, dominant form of transmission. The treatment, naturally, depends upon the possibilities of plastic surgery, individual for each case and determined by the deformity.

SPAETH, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

PRESENT STATUS OF FOCAL INFECTION. G. H. COLEMAN, J. A. M. A. **151**:280 (Jan. 24) 1953.

The important subject of focal infection has been brought up to date by Coleman, who has integrated the facts so that there is greater clarification now than ever before. Many years ago too much was claimed for the theory of focal infection. Skepticism resulted that led to much clinical and experimental research.

With continuous research by outstanding men in the medical and dental professions in various centers, the concept of focal infection once again has become an accepted principle in the practice of medicine and dentistry.

Focal infection and its effects as well as its treatment and prophylaxis from the medical and dental aspects are so thoroughly detailed in this short article that the reader is urged to consult it so that he may draw his own conclusions.

The specialty of otolaryngology has so much to do in this accepted principle that this revitalized concept will intensify the efforts of all otolaryngologists to continue their good work.

GORDON, Philadelphia.

PROBLEMS OF THE OTOLARYNGOLOGIST IN SURGERY OF THE NECK. J. G. GILBERT and L. J. FEIT, J. Internat. Coll. Surgeons **21**:93 (Jan.) 1954.

Gilbert and Feit point out that the early diagnosis of malignant disease of the aural, nasal, oral, and laryngeal cavities is the responsibility of the otolaryngologist. They discuss the anatomic aspects of the lymphatic system of the head and neck, the lymph node drainage of the head, and the various types of incisions in the neck (the latter with line drawings).

They have attempted a simplification and standardization of surgical procedures that have given them the best results, which procedures, they feel, are in the domain of the otolaryngologist.

GORDON, Philadelphia.

HISTAMINIC CEPHALALGIA (SUICIDE HEADACHE). SAM E. ROBERTS, Laryngoscope **64**:35 (Jan.) 1954.

Roberts describes a histamine cephalalgia as "a two phase unilateral agonizing headache, occurring mostly at night or when the patient is relaxed, and results from rapid dilation of the external carotid artery." The attacks are sudden and short and frequently recur three or four times a night. There is unilateral lacrimation, raving, and screaming. Sufferers frequently threaten to commit suicide. Desensitization to histamine gives excellent results.

HITSCHLER, Philadelphia.

EFFECT OF CHLORTETRACYCLINE (AUREOMYCIN) ON THE CLOTTING TIME OF BLOOD. HARRY SOROFF and STEPHAN ROSENAK, New York J. Med. **54**:93 (Jan. 1) 1954.

Soroff and Rosenak report from the Surgical Division of the Montefiore Hospital, New York City. In 1945 Muldovsky and Hasselbrook, for the first time, showed that penicillin, administered intramuscularly, reduced clotting time. Others confirmed this in laboratory animals. But in certain cases the clotting time has shown an increase when chlortetracycline (Aureomycin) was given! These variations are explained by two factors. One factor is emotional stress, which results in the outpouring of adrenaline. Innerfield says that the trauma of intravenous injection or cardiac puncture causes prolongation of the clotting time in apprehensive patients. But Soroff and Rosenak conclude that "there is no cumulative effect of aureomycin on the coagulation time of blood when it is given in doses of 250 mg. or less and in the accepted dosage schedule." Tables and figures are set down to show the variations in some 40 patients. The most significant evaluation occurred in 15 persons who received a single dose of chlortetracycline, resulting in a depressed coagulation time of more than one minute. This occurred in 83% of all patients. Three, or 8.5%, showed only slight depression of less than one minute. Three others showed a prolongation of coagulation time. Soroff and Rosenak state that no significant change in prothrombin time occurs after a single dose of chlortetracycline. However, after six doses "there is some tendency toward a prolongation of the prothrombin time."

ABSTRACTOR'S COMMENT: The most that can be said of these investigations is that they are inconclusive. Coagulation time may be either increased of diminished according to various factors in patients, the agent administered, and the dosages used. There seem to be no permanent effects one way or the other, which is, after all, the most important item.

I. W. VOORHEES, New York.

News and Comment

GENERAL NEWS

Specialists Certified by the American Board of Otolaryngology.—The following physicians were certified as specialists by the American Board of Otolaryngology at the recent examination held in Boston, May 18-20, 1954:

Alo, Joseph, Bronx, N. Y.
Baxter, James Douglas, Montreal, Canada.
Blanchard, Cyrus Lloyd, Ann Arbor, Mich.
Brackett, John Gordon, Atlanta.
Bradburn, Ian Muir, New Orleans.
Brown, John Joseph, Chelsea, Mass.
Bushnell, James Joseph, Mobile, Ala.
Campbell, Jack Swope, Battle Creek, Mich.
Ciell, August Paul, St. Albans 25, N. Y.
Couch, Ray Edwards, Port Arthur, Texas.
Cox, Lawson Glynn, Washington, D. C.
Cullum, Albert George, Jr., Fargo, N. D.
Duff, Robert L., McKinney, Texas.
Dwyer, Frank Philip, Jr., Catonsville, Md.
Farrior, Richard Thomas, U. S. Army, % PM, N. Y.
Fenning, Frederick Reid, Binghamton, N. Y.
Garlinghouse, Arnet John, Randolph Field, Texas.
Gupta, Indar Sagar, Boston.
Hasse, Frederick Robert, Drexel Hill, Pa.
Hays, Jack Ross, Mobile, Ala.
Hemenway, William Garth, Chicago 37.
Hui, Robert M., New York 32.
Jones, Arthur Denton, Jacksonville, Fla.
Kent, Charles, Boston.
Lagonegro, John Harry, Elmira, N. Y.
Leek, Joseph Hugh, Augusta, Ga.
Linthicum, Frederick H., Jr., Los Angeles.
Littell, Joseph J., Jr., San Rafael, Calif.
Livingood, William C., Philadelphia 45.
Maher, Joseph John, Sayre, Pa.
Marbry, George Walter, Cheyenne, Wyo.
Martin, Roy Allan, Louisville, Ky.
Petitti, Leonard, Colorado Springs, Colo.
Rice, Earle Winslow, Worchester, Mass.
Rothweiler, Robert J., St. Louis.
Rullan, Antonio, Santurce, Puerto Rico.
Samuelson, Arthur, Bridgeport, Conn.
Saunders, William H., Ann Arbor, Mich.
Serio, Joseph C., Buffalo, N. Y.
Stonkus, John A., Marysville, Calif.
Sudhoff, Max Frederick, Toledo 2, Ohio.
Weekes, Don James, Boston 15.
Wenger, Alvin, Baltimore.
Wilder, Raboteau Terrell, Bluefield, W. Va.
Yourd, Raymond Atchison, Champaign, Ill.
Zerne, Gustave Edward, West Chester, Pa.

The next examination of the Board will be held in the Waldorf-Astoria Hotel, New York City, Sept. 13-17, 1954. For information contact the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City.

JOINT MEETING OF THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS AND THE SOCIETY OF MILITARY OPHTHALMOLOGISTS

The Society of Military Otolaryngologists and the Society of Military Ophthalmologists will hold a joint dinner meeting at the time of the XVII International Congress of Ophthalmology and the annual meeting of the American Academy of Ophthalmology and Otolaryngology in New York City in September.

Cocktails and dinner will be served at 6 p. m. on Tuesday, Sept. 21, 1954, in the South and Center Lounges, Shelton Hotel, Lexington Ave. and 49th St., New York City (across the street from the Waldorf-Astoria).

All members, otolaryngologists and ophthalmologists on active duty with the Armed Forces, are invited to attend. Application should be made to Colonel J. H. King Jr., Secretary-Treasurer, Society of Military Ophthalmologists, Eye Clinic, Walter Reed Army Hospital, Washington 12, D. C.

SEVENTY-FIFTH ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

At the 75th Annual Meeting of the American Laryngological Association, held in Boston on May 27 and 28, 1954, two awards were presented. Dr. Louis H. Clerf, of Philadelphia, received the gold medal of the deRoaldes Award, and Dr. Henry B. Orton, of Newark, N. J., the Newcomb Award.

AWARD OF MERIT TO DR. STACY R. GUILD

At its annual meeting on May 23, 1954, the American Otological Society, Inc. awarded a Silver Medal, a suitably inscribed scroll, and an honorarium of one thousand dollars to Stacy R. Guild, Ph.D., of Baltimore. The award was made in recognition of the many contributions made by Dr. Guild in the field of research in otology during the past years.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations on the following days:
Sept. 13-17, 1954, in New York City, at the Waldorf-Astoria.
March 6-10, 1955, in Richmond, Va., at the Hotel Marshall.

INDEXES

This issue of the ARCHIVES appears in two parts, the second of which is the index for the preceding volume. To make the index more usable to the readers and to enable them to find information with less difficulty than has been encountered in previous volumes, the index has been broken down into three separate indexes: Title Index, Author Index, and Subject Index. The Title Index, which will take the place of the six-month Table of Contents, previously published with the index of the journals, has been prepared in such form that it can be separated from the other indexes and bound in the front of the volume. It consists of the titles of the articles arranged in alphabetical order and accompanied by the name of the author, the name of the senior author if there are more than two, the names of both authors if there are two, and the page number on which the article begins. The Author Index contains the names of all authors of articles appearing in the ARCHIVES and of the presenters of cases or papers before societies. The third index, the Subject Index, contains in alphabetical order the important subjects which have been dealt with in the volume. Individual abstracts are no longer indexed; only the categories under which they have been classified are listed. Book Notices and news items are included in this Index.

Directory of Otolaryngologic Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont.

International Bronchoesophagological Society

President: Dr. André Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Third International Congress of Bronchoesophagology.
Place: Lisbon, Portugal. Time: Oct. 10-13, 1954.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: United States of America. Time: 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary: Dr. Hugh A. R. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Walter H. Theobald, 307 N. Michigan Blvd., Chicago 1.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN BOARD OF OTOLARYNGOLOGY

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Statler Hotel, Boston.
Examinations: Waldorf-Astoria Hotel, New York, Sept. 13-17, 1954; Hotel Marshall, Richmond, Va., March 6-10, 1955.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3.
Place: Statler Hotel, Boston.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Henry B. Orton, 224 Delavan Ave., Newark 4, N. J.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Hollywood Beach Hotel, Hollywood, Fla. Time: March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
President-Elect: Dr. Kenneth M. Day, 121 University Place, Pittsburgh.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Statler Hotel, Boston.

SECTIONS:

Eastern.—Chairman: Dr. Daniel S. Cunning, 118 E. 53rd St., N. Y. 22.
Place: Hotel Syracuse, Syracuse, N. Y.
Southern.—Chairman: Dr. William C. Wolfe, 332 W. Broadway, Louisville 2.
Place: Andrew Jackson Hotel, Nashville, Tenn.
Middle.—Chairman: Dr. French K. Hansel, 634 N. Grand Blvd., St. Louis 3.
Place: The Drake, Chicago.
Western.—Chairman: Dr. Guy L. Boyden, 1735 N. Wheeler Ave., Portland 12, Ore.
Place: The Elks Club, Los Angeles.

AMERICAN OTOLOGICAL SOCIETY, INC.

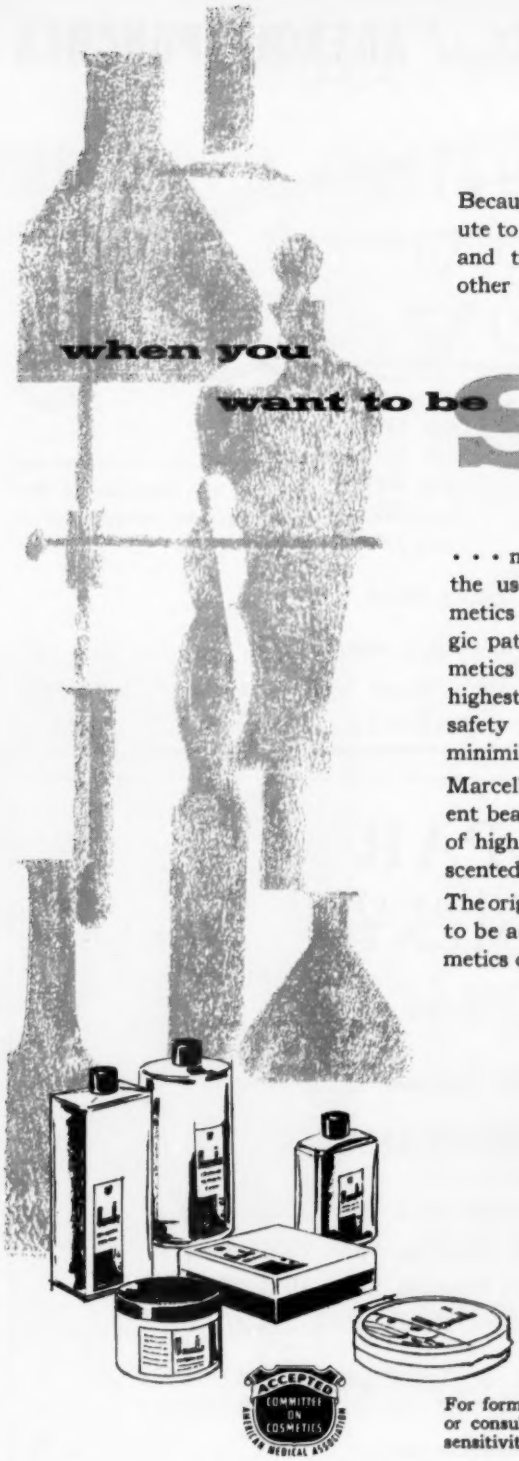
President: Dr. Staunton Wishart, 170 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37.
Place: Hollywood Beach Hotel, Hollywood, Fla. Time: March 17-18, 1955.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Harry Neivert, 555 Park Ave., New York 21.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
President-Elect: Dr. Albert D. Reudemann, 1633 David Whitney Building, Detroit 26.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.



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Archives of Otolaryngology, Sept. 1952: 241-249
The Eye, Ear, Nose & Throat Monthly, April, 1950: 200-203
Bulletin of the Johns Hopkins Hospital, Sept., 1948: 225-228
Archives of Otolaryngology, March, 1947: 294-297

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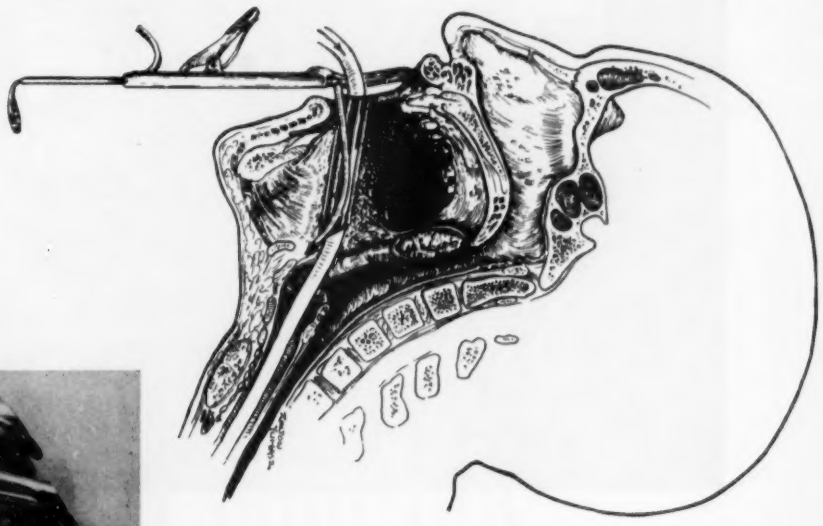

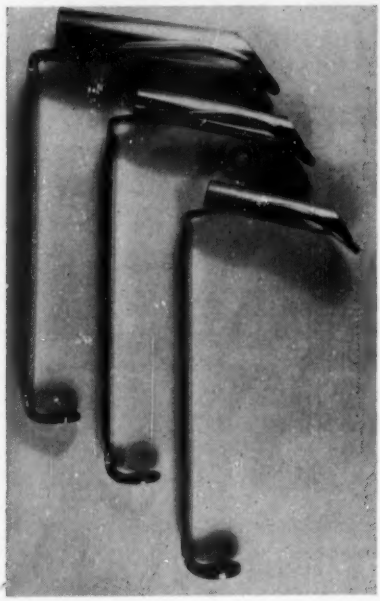

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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(1) Douglass, C. C.: Laryngoscope 58: 1274, 1948; (2) Anderson, J. R., and Steele, C. H.: Laryngoscope 58: 1279, 1948; (3) Long, P. H.: A-B-C's of Sulfonamide and Antibiotic Therapy, Philadelphia, W. B. Saunders, 1948, p. 152.

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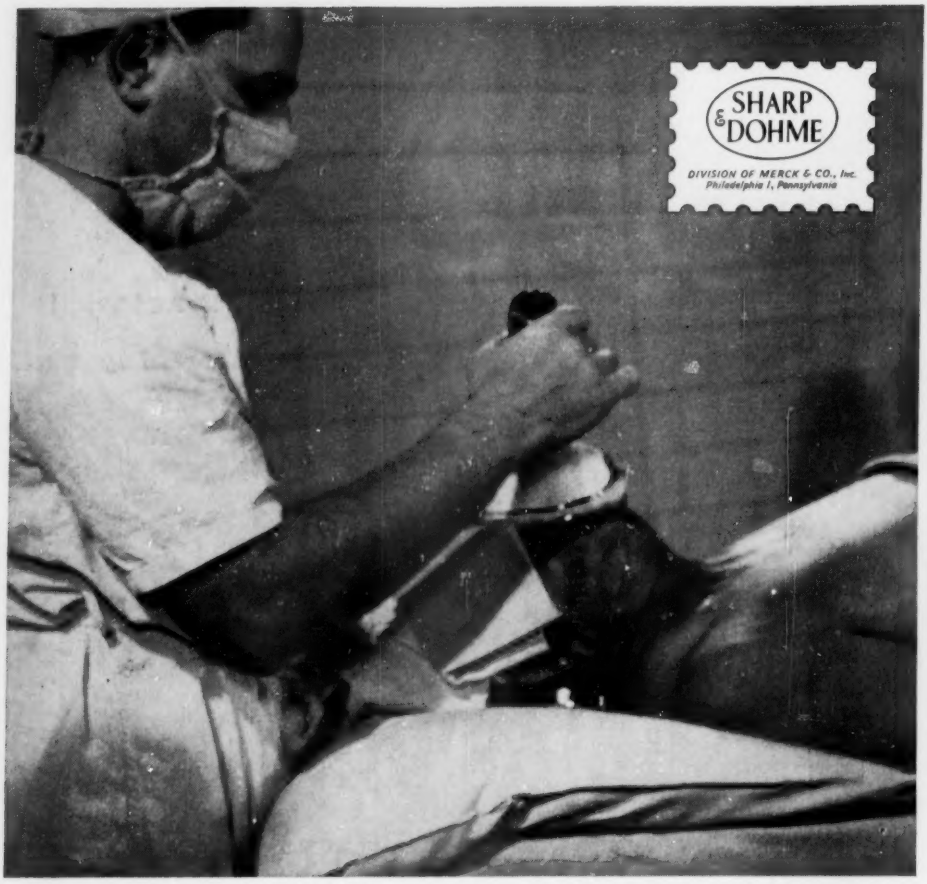

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